Dissertation on

A STUDY ON OPHTHALMIC MANIFESTATIONS OF MIGRAINE

Submitted in partial fulfilment of requirements of

M.S. OPHTHALMOLOGY

BRANCH-III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI-600003

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL – 2016

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON OPHTHALMIC MANIFESTATIONS OF MIGRAINE"** is a bonafide record of the research work done by **Dr.Krishnan R ,** Post graduate in Regional Institute of Ophthalmology, Madras Medical College and, Government General Hospital, Chennai-03. In partial fulfilment of the regulations laid down by The Tamil Nadu Dr.M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2012-2015

Dr. K. Namitha Bhuvaneswari, M.S.,DO.
Chief, Cornea & contact lens services
Regional Institute of Ophthalomology
Government Ophthalmic Hospital,
Egmore, Chennai - 600 008.

Dr.K.Namitha Bhuvaneswari,M.S.,DO.
Director & Superintendent
Regional institute of Ophthalmology
Government Ophthalmic Hospital
Egmore, Chennai-600008

Dr.R.Vimala M.D.
Dean,
Madras Medical College
And Government General Hospital
Chennai-600003

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I wish to express my sincere thanks to all my professors, assistant professors and all my colleagues who had helped me in bringing out this study.

Finally, I am indebted to all the patients for their sincere co-operation for the completion of this study.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"A STUDY ON OPHTHALMIC MANIFESTATIONS OF MIGRAINE"** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr. K. NAMITHA BHUVANESWARI M.S.,D.O.**

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CERTIFICATE OF APPROVAL

To
Dr.Krishnan R
Postgradaute M.S.(Ophthalmology)
Madras Medical College
Chennai 600 003

Dear Dr.Krishnan R,

The Institutional Ethics Committee has considered your request and approved your study titled **"A study on Ophthalmic manifestations of Migraine" No.14062015.**

The following members of Ethics Committee were present in the meeting held on 09.06.2015 conducted at Madras Medical College, Chennai-3.

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2. Prof.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
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13. Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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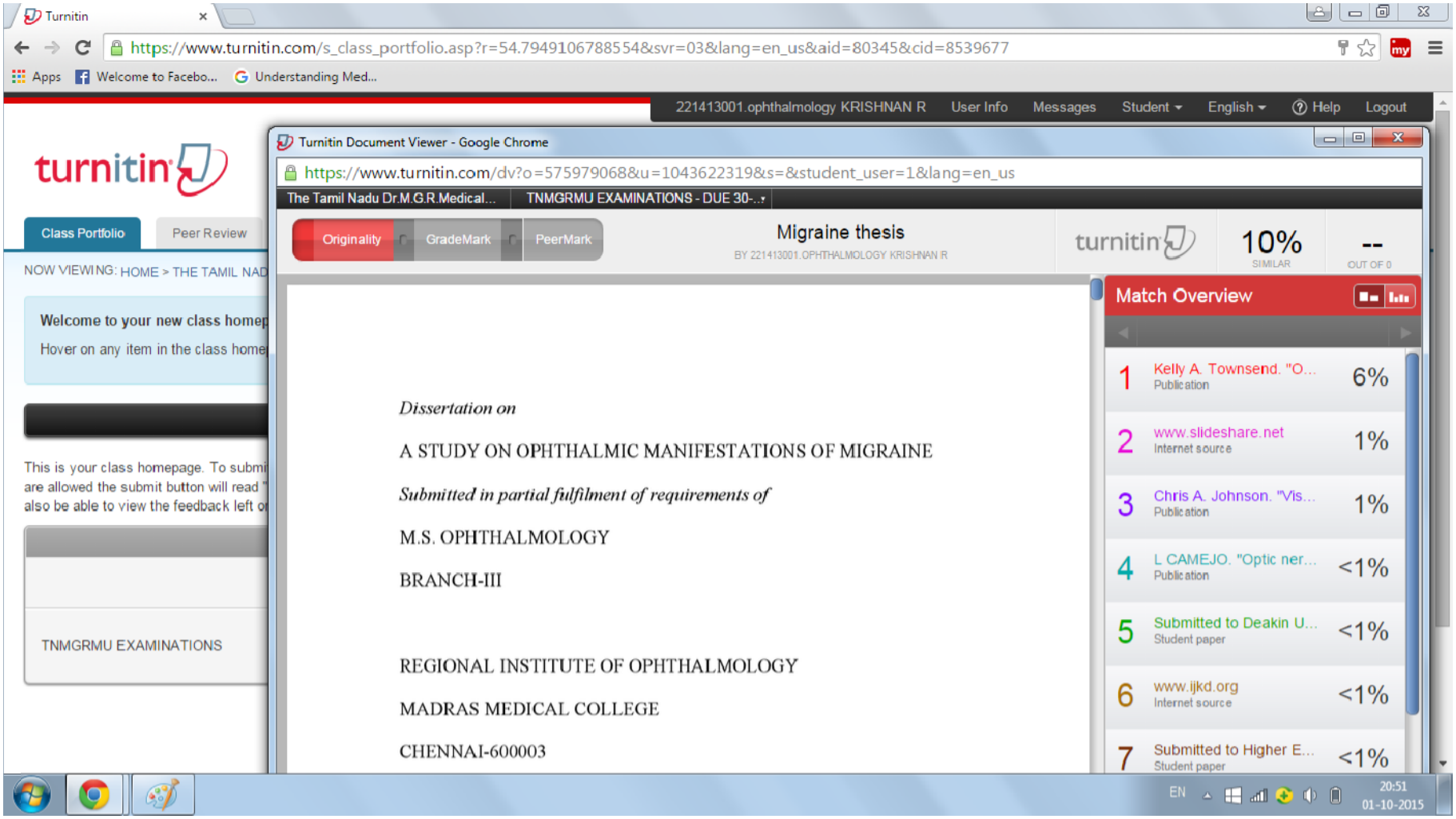
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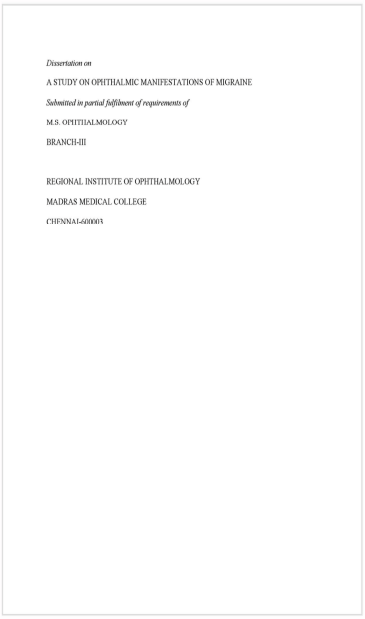

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CONTENTS

ABSTRACT

TOPIC

A study on ophthalmic manifestations of migraine

AIMS AND OBJECTIVES

1.To elucidate the various ophthalmic manifestations of different types of migraine

2.To assess the response to treatment (with steroids) in ophthalmoplegic migraine.

3.To assess the visual field changes in migraine

4.OCT changes in migraine

5.To find out any association with other ocular disorders.

INCLUSION CRITERIA

1.Patients with migraine (migraine with aura and migraine without aura).

2.Retinal migraine

3.Ophthalmoplegic migraine

EXCLUSION CRITERIA

Any other type of headache other than migraine,refractive errors,head or eye trauma,

eyes with retinal or optic disc pathology,cataract,corneal opacity,ocular laser treatment,CNS

disorders like tumors,infarcts,diabetic and hypertensive retinopathy

AGE:5 to 60 years

STUDY DESIGN:Prospective clinical study

NUMBER OF PATIENTS:50

CONCLUSION

The mean age of presentation was 40.58 years of age.Females were more commonly affected.Visual phenomena associated with migraine was present in 30% of patients with migraine.Photophobia in 18% of patients. RNFL thinning was noted by OCT in BE- 50% of patients(single quadrant thinning),RE:8% of patients(single quadrant thinning),LE:8% of the patients(single quadrant thinning),multiple quadrant thinning:2% of patients. Patients with ophthalmoplegic migraine responded to treatment with steroids and completely recovered in 2 months.This proved that ophthalmoplegic migraine is an inflammatory demyelinating neuropathy.The mean age of presentation was 5 years of age in our study.

PART 1

GENETICS

Several factors allude to a genetic basis for migraine based on twin studies,family studies of rare genetic variations of migraine.Monozygotic twin studies have a higher concordance rate for migraine than dizygotic twin studies.Genes have been identified in some patients with rare forms of migraine such as familial hemiplegic migraine(CACNA1A subunit on chromosome 19p13). Overall there is no specific pattern of inheritance and multifactorial inheritance(environmental and genetic) plays a role.

MECHANISM

The aura symptoms of migraine are likely caused by cortical spreading depression(CSD).CSD involves initial neuronal hyperpolarisation.During this time positive symptoms occur and there is increased regional cerebral blood flow.The wave of hyperpolarisation is followed by depolarisation,leading to negative aura symptoms.Aura symptoms are produced in relation to the areas of cerebral cortex affected.

Primary and visual association areas : Visual symptoms

Parietal cortex: Somatosensory symptoms

Motor cortex: Weakness

The dynamic nature of CSD correlates with the speed of scintillating scotoma moving across the visual field and explains the typical march of migraine aura symptoms.

PATHOPHYSIOLOGY

The pain of migraine is linked to the pain sensitive structures in the brain, the dura and cranial blood vessels. These structures are innervated by the first division of trigeminal nerve(ophthalmic) anteriorly and branches of C1 and C2 posteriorly. The interaction among the trigeminal nerve, autonomic nerves and blood vessels is referred to as trigeminovascular system. The central trigeminovascular neurons receive multiple sensory inputs from the meninges and the periorbital tissues. When activated by meningeal nociceptors, the central neurons may misinterpret the pain as originating in or near the eye causing the referred periorbital pain of migraine.

When neurons are activated, inflammatory molecules(substance P,calcitonin gene related peptide, vasoactive intestinal peptide) are released near meningeal blood vessels, activating meningeal nociceptors and producing vasodilation,plasma extravasation, mast cell degranulation and platelet aggregation. This creates a condition of sterile inflammation around dural blood vessels. Within 5-20 min of a migraine headache, peripheral nociceptors are sensitised, producing a burst of action potentials in response to cerebrospinal fluid pulsations and leading to throbbing pain. Between 20 minutes and 2 hours after a migraine attack central neurons are sensitised and produce a heightened response to otherwise innocuous stimuli.This phenomenon, cutaneous allodynia, accounts for facial and scalp tenderness that occurs during migraine

PATHOPHYSIOLOGY OF ASSOCIATED SYMPTOMS

Brain structures such as dorsal raphe nucleus, nucleus raphe magnus and trigeminal nucleus caudalis project to various areas of the brain and spinal cord to further modulate the pain and other symptoms of migraine. Connections through the superior salivatory nucleus to the pterygopalatine ganglion may be responsible for ocular autonomic features of migraine such as lacrimation, lid edema and conjunctival injection. Hypothalamic projections may mediate the episodic pattern of attacks in some headache disorders as well as changes in arousal. Input to the frontal cortex may lead to difficulty concentrating and emotional changes associated with migraine. Migraine pain is modulated through the thalamus.The trigeminal nucleus caudalis and dorsal horn of C1 and C2 also contribute to the pain of migraine particularly posterior head and neck.

CLINICAL FEATURES

Migraine may begin at any age and is not rare in infancy and early childhood. Additionally the character of migraine often changes throughout life. Women commonly experience a shift in their migraine pattern during times of hormonal fluctuation, such as menarche, pregnancy and menopause.

Patients who experienced migraine pattern with aura in adolescence or young adulthood may have improvement in headaches but experience recurrent aura symptoms as they get older.

A fully developed migraine attack can be divided into four phases,each of which may have ophthalmic manifestations:

Premonitory phase:

Symptoms occurring hours to days before a migraine attack that may include depression, sleep disorders, hypoactivity or arousal, abdominal discomfort, food carvings, epiphora, dulled or heightened mentation, uncontrollable yawning, heightened perceptions of taste and smell and visual difficulties.

Aura:

Focal neurological symptoms immediately preceding and sometimes accompanying the headache. It is present only in 20% of the patients.

Headache:

mild, moderate, severe

Postdrome:

Post headache alteration of mood or mentation, difficulty concentrating including drowsiness, lethargy or epiphora. Symptoms may last upto 24 hours.

The most common symptom of migraine is pulsatile pain present in 85 % of patients followed by photophobia(76%) and nausea(74%).

Hemicranial pain occurs in 59% of patients.

Worsening of headache with physical activity is characteristic of migraine which distinguishes from other types of headache.

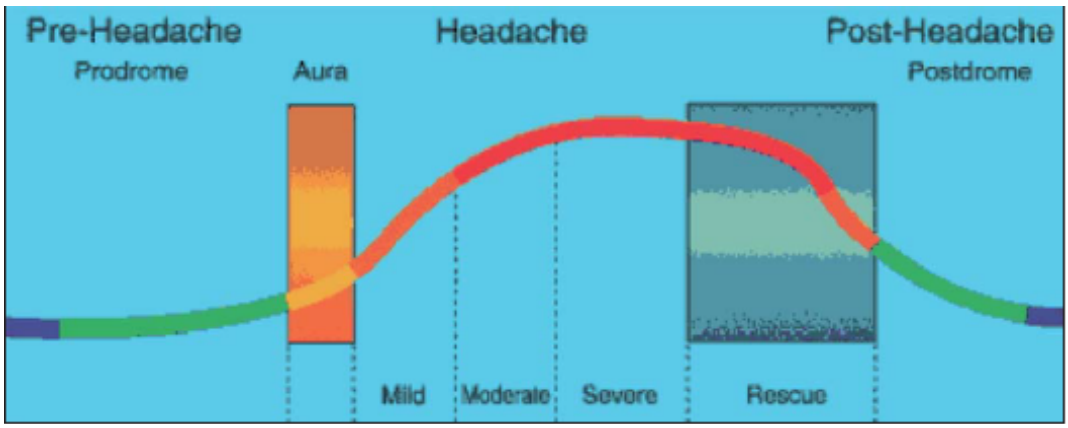


Figure 1:Phases of migraine

OPHTHALMIC SYMPTOMS & SIGNS:

Typically ophthalmic features of migraine occur during the aura phase but they may occur at any time during the migraine period. Blurred vision, a non specific feature of migraine occurs in 40% of patients at some point of time during migraine. The major types of ophthalmic symptoms are positive symptoms, negative symptoms, autonomic manifestations and efferent symptoms or signs

POSITIVE SYMPTOMS

Seeing shapes or lights that are not present is the most typical manifestation of migraine aura. The characteristic scintillation scotoma with fortification spectra is virtually pathognomic of migraine. It generally begins as a paracentral shimmering effect that enlarges and expands over the hemifield. The illusion may be colourful or silver and black. The periphery takes on a jagged or herring bone pattern that shimmers or scintillates. An area of relative or absolute scotoma is frequently observed within the confines of the shimmering border. The rate of progression of the scintillating scotoma across the visual field corresponds to the rate of cortical spreading depression across the visual cortex 3mm/min. This symptom usually lasts

10 – 30 min and frequently heralds a headache attack. Other positive symptoms include light flashes, sparkles, kaleidoscope vision, fragmented vision(like cracked glass) and a sensation of heat waves.

Cortical visual disturbances (similar to those of epilepsy) include metamorphopsia, micropsia or macropsia, Alice in Wonderland syndrome, mosaic vision**.**

NEGATIVE SYMPTOMS

Homonymous hemianopic or quadrantanopic visual field defect.Tunnel vision sometimes progressing to complete blindness may occur.

AUTONOMIC FEATURES

These features define trigeminal autonomic cephalgias during a migraine attack. Horners syndrome, conjunctival injection, mild ptosis and eyelid edema may accompany nasal congestion or rhinorrhea. The nasal symptoms often lead to erroneous diagnosis of sinus headache. Transient anisocoria may be a part of migraine attack either in isolation or with an oculomotor nerve palsy or Horners syndrome

OTHER NEUROLOGICAL SYMPTOMS

These features are much less common and may be a part of aura or a manifestation of basilar type of migraine. Diplopia may be a symptom of basilar type of migraine (Bickerstaffs migraine) which often affects adolescent girls but may affect both males and females of all ages.Other symptoms of this uncommon

migraine are vertigo, tinnitus, hearing impairment, ataxia, impaired cognition and altered level of consciousness. Bilateral symptoms and signs are typical such as paresthesias and numbness.

OTHER FEATURES

Photosensitivity, persistent positive visual phenomena ("visual snow"), difficulty tolerating a busy environment, cortical blindness, visual distortion("heat off the pavement"), isolated papillary mydriasis

MIGRAINE VARIANTS

RETINAL MIGRAINE(OCULAR MIGRAINE)

Ocular (retinal) migraine consists of monocular visual symptoms with or without associated headache. The terminology is confusing and it is often mistakenly used to describe a visual aura of cortical origin or the presence of binocular aura(eg., homonymous hemianopia) without headache. When obtaining the description of symptoms from the patient, it is helpful to ask what they observed when they covered or closed either eye. In case of visual loss, an accounting of their vision when looking straight ahead with both eyes open usually distinguishes monocular from binocular visual loss.

The International Headache Society classification specifies atleast two reversible attacks lasting less than 60 minutes associated with migraine headache. Visual symptoms may be positive or negative. When headache occurs, the visual loss is generally ipsilateral to the pain..

Young women are frequently affected. It is controversial whether it is pathophysiologically related to more typical migraine. Unilateral visual loss which is transient in nature may occur. This may resemble amaurosis fugax from thromboembolic disease.

These spells are generally painless and thus do not meet strict criteria for ocular migraine. They last seconds to hours ,usually 5-45 min and are described as a monocular shade, curtain, or tunnel vision. The spells often come in a flurry in the sixth or seventh decade. Many patients have a prior history of migraine headaches but these late life episodes may occur in previously asymptomatic patients.

ASSOCIATIONS

Narrowing or occlusion of retinal arteries or veins has been observed in some cases. Ischaemic optic neuropathy, retinal vein occlusion, retinal or optic nerve haemorrhages may be noted in some cases. Although visual loss during ocular migraine is generally reversible, permanent visual loss sometimes occurs. Thus preventive treatment with calcium channel blockers ,low dose aspirin or other migraine prophylactic medications may be warranted

BENIGN EPISODIC PUPILLARY MYDRIASIS

It is an uncommon condition characterised by unilateral episodic mydriasis lasting minutes to a week. It may be associated with headache or orbital pain but is frequently painless. It is likely a migraine variant since over 50% of patients have a history of migraine. The involved pupil is usually poorly reactive or unreactive to light and near stimuli, and does not constrict with pilocarpine. Differential diagnosis includes Adies tonic pupil, pharmacologic mydriasis and early oculomotor nerve compression.

OPHTHALMOPLEGIC MIGRAINE

COMMON AGE GROUP AFFECTED: paediatric age group, commonly 5 to 8 yrs

INCIDENCE:0.9/1,000,000

Ophthalmoplegic migraine is an ocular disorder which is characterised by ophthalmoplegia following many episodes of migraine. The disorder always begin in childhood. Isolated Oculomotor nerve palsy is common. Pupil involvement is the rule. Abducent and trochlear nerve palsies are rare. Prognosis is good as symptoms usually resolve.

It is not a true migraine so removed from International Headache Society classification of migraine. Headache resolves over a few days but cranial neuropathy may take weeks to resolve. It may continue to adulthood.

PATHOPHYSIOLOGY

Earlier it was thought that during the attack, carotid artery wall becomes edematous. This compresses the oculomotor nerve in the cavernous sinus and causes third nerve palsy. The latest finding is that ophthalmoplegic migraine is an inflammatory demyelinating neuropathy of third nerve which also irritates the fifth nrve roots present in the same nerve. GdMRI proves that the oculomotor nerve becomes reversibly enhanced, especially the cisternal portion.

CLINICAL FEATURES

Usually patients complain of headache, drooping of upper lid, double vision (if no drooping) of sudden onset for short duration of about a week or 10 days.

Headache is usually more in the frontal region and is usually associated with projectile vomiting and head banging which suggestive of temper tantrums. There is intolerance to noisy environment and headache is usually throbbing in nature. There is usually a past history of previous migraine headaches for which the patient would have taken medications. There will be associated 3rd(most common),4th or 6th cranial nerve palsies.

DIFERENTIAL DIAGNOSIS

1. Aneurysm
2. Infections(eg.,Lyme disease,syphilis,HIV)
3. Microvascular cranial mononeuropathy
4. Lymphoma
5. Leukemia
6. Tolasa Hunt Syndrome
7. Sarcoidosis
8. Sphenoid sinusitis

MRI FINDING

Enhancement of third nerve on MRI is frequently observed.

INVESTIGATIONS

1. Baseline investigations like complete blood count(Hb,TC,DC,platelet count, PCV), blood urea , serum creatinine, serum sodium. potassium, SGOT, SGPT, X ray chest.
2. Vasculitis work up:ESR,CRP.ANA,ANCA,RF.
3. Thyroid profile:T3,T4,TSH.
4. Lumbar puncture and CSF analysis.

5. Metabolic parameters like serum lactate,ammonia,urine porhyrin.

6. Complement factors.

7. ECG,echocardiogram.

8. MRI.

MANAGEMENT

Medications for migraine (eg.,T.Propranalol).

Antidepressants like T. Amitryptilline

Steroids(1 mg/kg bodyweight) and slowly tapered.

Complete recovery is the rule

ASSOCIATED VISUAL FEATURES OF MIGRAINE

VISUAL SNOW

Visual snow is the most perplexing and frustrating condition characterized by persistent positive visual phenomena which most commonly occurs in migraineurs. Patients describe snow, ants dots or rain continuously encompassing the entire visual field. Complex illusions have also been reported. Visual snow frequently occurs in high functioning, observant individuals. They are able to see through the static but it is distracting and annoying. It may persist for many years. Neuro-imaging studies are unrevealing and treatment is usually unsatisfactory. The pathophysiology is uncertain but spontaneous cortical discharges, persistent hyperexcitability of the visual cortex and a heightened awareness of normal visual phenomena are postulated.

PHOTOPHOBIA

Photophobia is an abnormal sensitivity to light which is one of the diagnostic criteria for migraine. It is present in 80% of patients during a migraine headache. It may also be a part of the aura or premonitory symptom of migraine. Additionally patients with migraine have more light sensitivity than control patients in between attacks.

Photosensitivity is the visual system analog of allodynia which occurs in other neurological sensory systems during migraine (allodynia is the painful perception of a stimulus that is generally not painful).Photophobia often occurs during a migraine attack particularly chronic cases. As with other forms of allodynia the stimulus may also be a migraine trigger. Bright lighting such as sunlight, fluorescent light, bright snow or flickering lights triggers migraine attacks in 30-60% of migraineurs. Many patients also have a heightened sensitivity to complex visual patterns or a busy visual environment in between attacks. Sympathetic stimulation and down regulation of visual thresholds by the trigeminal nerve usually occurs.

VISUAL PHENOMENA ASSOCIATED WITH MIGRAINE

FORTIFICATION SPECTRA

Arc of jagged, serrated or zigzag lines. They are also caused by occipital AVM, seizures and space occupying lesions.

SCINTILLATING SCOTOMA

Positive fortification spectra on outside and negative scotoma in the middle. They are also caused by seizures, occipital AVM, and space occupying lesions.

MICROPSIA

Objects appear too small. It is also caused by seizures, occipital AVM, space occupying lesion and macular disease.

MACROPSIA

Objects appear large. It is also seen in seizures, occipital AVM, and space occupying lesion.

METAMORPHOPSIA

Objects appear distorted in shape. It is also seen in macular disease and seizures.

“ALICE IN WONDERLAND SYNDROME”

Episodes of distorted body image. It is also seen in seizures, occipital AVM, and space occupying lesions.

PALLINOPSIA

Persistence of visual images. Also seen in seizures,parieto occipital damage and some medications.

CEREBRAL POLYOPIA

Perception of multiple images in both eyes. Also seen in seizures and right occipital damage

ACHROMATOPSIA

Inability to perceive colour. Also seen in bilateral occipito temporal damage.

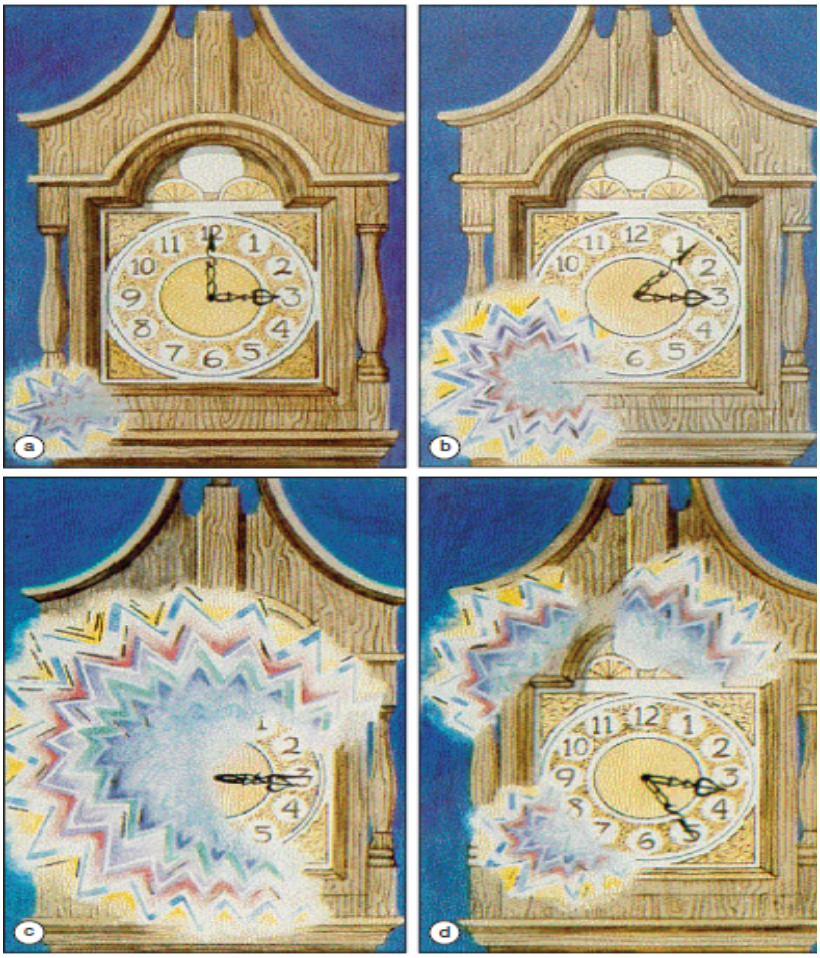


Figure 2 : **Scintillation scotoma which is the characteristic visual feature of migraine.**

Figure 2a : **Scintillation scotoma initially appears as a small paracentral visual disturbance**

Figure 2b&c : **Scintillation scotoma then starts expanding over one hemifield**

Figure 2d : **Scintillation scotoma then breaks up in the periphery**

OTHER TYPES OF MIGRAINE

MIGRAINE ASSOCIATED WITH HEMIPLEGIA

This type of migraine is recurrent and familial.It is associated with aura and motor weakness.

INHERITANCE:

Autosomal dominant with variable penetrance

ONSET:

Typically in childhood and nearly always by the age of 30 yrs

GENETICS:

Familial hemiplegic migraine type 1 localises to chromosome 19 and type 2 is localized to chromosome 1.

CLINICAL FEATURES:

1. Typical aura
2. Disturbance of consciousness
3. Fever
4. CSF pleocytosis
5. Confusion during an attack
6. MRI brain is normal

TREATMENT

Differs from other forms of migraine. Medications with vasoconstrictor effects are best avoided. Treatment is usually with intravenous dopamine agonists, calcium channel blockers and acetazolamide.

BASILAR TYPE OF MIGRAINE

Migraine is recurrent.Aura is usually present.Weakness is absent.Cerebral hemispheres or medulla or pons is involved.It is usually rare and should only be diagnosed after vertebrobasilar ischaemia has been ruled out.

TENSION TYPE HEADACHE(TTH)

TTH is an important differential diagnosis for migraine. Migraine and TTH may be found in the same patient. Muscle tension is often considered as a unique feature of TTH, while migraine is commonly felt not to be associated with muscle tension or neck pain.

CLASSIFICATION

Episodic TTH and chronic TTH

EPISODIC TTH

Episodic TTH is characterized by headache which occurs frequently. Headache is usually bilateral. Headache is not related to routine physical activity. There is no nausea or vomiting. Photophobia or phonophobia may occur. The headaches occur fewer than 15 days a month for atleast 3 months.

CHRONIC TTH

In chronic TTH, the frequency of headache is increased. They occur greater than 15 days a month. This usually happens for a minimum of three months continuously.

PATHOPHYSIOLOGY

TTH occurs due to sustained contraction of pericranial muscles. Peripheral pain mechanisms act as a trigger for a central process. Peripheral mechanisms may be most important in episodic TTH while in chronic TTH, central mechanisms may predominate.

TREATMENT

Acute therapy typically involve the structured use of simple analgesics (acetaminophen, aspirin),alone or in combination with NSAIDS. Overuse of these drugs must be limited. Prophylactic therapy must be considered when the frequency and duration of the headache is prolonged and over use is suspected. Tricyclic anti-depressants can be used for prophylactic therapy.

INVESTIGATIONS AND CLINICAL WORK UP

HISTORY

Detailed history taking is carried out in evaluating the ophthalmic features of migraine. History of diurnal variations, presence or absence of aura is asked. History of any associated symptoms like nausea, vomiting is asked .Patients with ophthalmoplegic migraine are carefully evaluated. As these patients are children, parents or guardians from whom the history taking is reliable is selected for the

study. Drug and treatment history is asked and duration of illness is also one of the most important parameters to be asked for.Triggers of migraine are

1.Emotional stress

2.Hormonal changes in women

3.Weather

4.Sleep disturbances

5.Odours

6.Lights

7.Alcohol

8.Smoking

9.Sleeping late

10.Heat

11.Food

12.Exercise

Past history of diabetes, hypertension, bronchial asthma,COPD,coronary artery disease, tuberculosis, renal disorder, thyroid disorder drug abuse is important. Personal history of smoking, alcoholism, diet intake is asked. Family history of migraine is asked.

GENERAL EXAMINATION AND INVESTIGATIONS

Pulse rate, BP, respiratory rate, temperature are recorded. Systemic examination of cardiovascular system, respiratory system, abdomen, central nervous system is done.General investigations like blood sugar, hemoglobin, total count, differential count, lipid profile, ESR done.

OPHTHALMIC INVESTIGATIONS

VISUAL ACUITY

Visual acuity is tested using snellens visual acuity charts

COLOUR VISION

Colour vision is a perceptual phenomenon not just a physical property of an object. There are three different types of cones: red sensitive (erythrolabe),green sensitive (chlorolabe) and blue sensitive (cyanolabe),which combinedly perform the function of colour vision. All colours are a result of admixture in different proportion of the three primary colours:

1.Red(723-647 nm)

2.Green(575-492 nm)

3.Blue(492-450 nm)

Colours have 3 attributes:hue, intensity and saturation.

A normal person can see all wavelengths between violet to red. If the wavelength is shorter than that of violet, the light becomes ultraviolet(UV) and is beyond visibility. If the wavelength is greater than 750 nm,the light is infrared and is again beyond visibility. Human beings could have seen even UV light as blue cones retain some sensitivity at around 10 nm, but crystalline lens blocks all UV rays. Consequently after cataract surgery one can see UV rays to an extent.

The photochemical changes in the cone pigments followed by a cascade of biochemical changes produce a visual signal in the form of “cone receptor potential”. Cone receptor potential has a sharp onset but slow offset. It is transmitted to the other cells of retina across the synapses of photoreceptors, bipolar cells and horizontal cells. Then it is transmitted across the synapses of bipolar cells,ganglion cells and amacrine cells.

Trichromatic colour vision mechanism extends 20 – 30 degrees from the point of fixation. Peripheral to this red and green becomes indistinguishable and in the far periphery all colour sense is lost, although cones are still found in the region of retina.

Hue is the dominant spectral colour as determined by the wavelength of the particular colour. The lightness or brightness of the colour depends upon the luminosity of the component wavelength. In photopic vision(day light),normal eye has a peak luminosity function at approximately 555 nm and in scotopic vision(dim light) at about 507 nm. This wavelength shift is called “Purkinje shift”.Saturation refers to degree of freedom from dilution with white.It can be estimated by measuring how much of wavelength must be added to white before it is distinguishable from white. The more the wavelength to be added to make the discrimination, the lesser the saturation and vice versa.

TESTS FOR COLOUR VISION IN MIGRAINE

A chromatopsia is a visual phenomenon associated with migraine.Ishihara’s colour vision plates are used in RIOGOH for testing colour vision of the patients

REFRACTION

Refraction should be done in all patients with migraine.If the patients are above 40 years, full presbyopic correction should be given.Retinoscopic refraction done for all patients to rule out refractive errors as headache may be because of refractive errors.

Drug used is

1-5 years:1% atropine eye drops.

5-8 years:2% homatropine eye drops.

8-20 years:1 % cyclopentolate hydrochloride eye drops.

Above 20 years:0.5% or 0.8% tropicamide eyedrops.

SLIT LAMP BIOMICROSCOPIC EXAMINATION

Slit lamp examination is done for all patients with migraine. It helps to rule out anterior segment diseases of the eye like conjunctivitis, lid disorders(blepharitis, meibomitis), keratitis,uveitis ,cataracts.

INTRAOCULAR PRESSURE

Measurement of intraocular pressure is one of the routine investigations in migraine. It also helps us to find whether migraine is associated with other ocular disorders like glaucoma. Goldmann’s applanation tonometer is the gold standard in measuring intraocular pressure.

FUNDUS EXAMINATION

Fundus examination should be done in all patients with migraine. All patients are dilated using 0.8% Tropicamide eye drops and detailed fundus examination should be done. The following points are given importance while performing the fundus examination:

1.Disc changes:

The disc should be examined for its size, shape, colour, margins and cup-disc ratio. This is helpful to rule out other ocular disorders like glaucoma.

2.RNFL changes:

RNFL layer changes in all quadrants (superior, inferior, nasal and temporal) can be found out clinically using red free filter provided in the direct ophthalmoscope. Its helpful in assessing the RNFL layer changes in migraine

3.Macula:

Macula is examined using direct,indirect and slit lamp biomicroscopy with 90D lens. It is to rule out any other macular disorders like degenerations, holes, epiretinal membranes or dystrophies.

4.Peripheries:

The peripheral retina is examined using indirect ophthalmoscopy with 20D lens. This is done to rule out peripheral retinal diseases like retinal degenerations, detachments, breaks or holes.

GONIOSCOPY

Gonioscopy is used to study the angle structures of the eye. Because of total internal reflection, angle structures of the eye cannot be visualized by naked eye. So we use a gonioscopic lens to visualize the angle structures. It helps us to find whether the angles are open or closed. Thereby we can also find whether migraine is associated with ocular disorders like glaucoma. Direct or indirect lens can be used. Indirect lens helps us to grade the opposite angles and direct lens helps us to grade the angles in the same quadrant. Indirect lens may or may not use a coupling fluid.

SHAFFERS GRADING OF ANGLES

GRADE	STATUS	ANGLE STRUCTURES VISIBLE
4	Wide open (40 degrees)	Ciliary body band, scleral spur, trabecular meshwork, Shwalbe's line
3	Open (30 degrees)	Scleral spur, trabecular meshwork, Shwalbes line
2	Moderately narrow (20 degrees)	Trabecular meshwork, Shwalbe's line
1	Narrow (10 degrees)	Shwalbe's line only
0	Closed (0 degrees)	No angle structures visible

AUTOMATED PERIMETRY

Standard Automated Perimetry using an adaptive forecasting threshold technology for central visual field remains the preferred visual field testing method. Neuro-ophthal fields for migraine can also be evaluated using automated perimetry. It tells us about the visual field changes in migraine and also whether migraine is associated with other disorders like glaucoma.

Automated tests utilizes computer programs to vary test speed, target size and luminance. They also benefit from standardized testing conditions which can be used to interpret results across machines. They present light stimuli of varying luminance to the patient on specific locations for a given duration of time before the next stimuli is presented.

Both static and kinetic testing can be performed manually. Manual kinetic perimetry is more flexible and interactive for the patient and it provides the opportunity to evaluate the peripheral visual fields.

PROGRAM TESTS:

Test procedures to evaluate the macular region ,the central 30 degrees radius and the far peripheral visual field beyond 30 degrees are available. A variety of target sizes, durations and test presentation patterns can be selected.

OPTICAL COHERENCE TOMOGRAPHY(OCT)

The three dimensional structure of the optic nerve head and the peripapillary thickness of the retinal nerve fibre layer(significant in migraine) can be assessed quantitatively with precision using OCT.

OCT is a high resolution cross sectional imaging of the RNFL.It measures the intensity and echo time delay of back scattered and back reflected light from the scanned tissues.

PRINCIPLE :

Based on principle of Michelsons interferometry where low coherence infrared light is coupled to a fibre optic travels through a beam splitter and is directed through the ocular media to the retina and a reference mirror. The distance between the beam splitter and the reference mirror is continuously varied. When the distance between the light source and the retinal tissue = distance between the light source and reference mirror, the reflected light interacts to produce an interference pattern. The interference is measured by a photodetector and processed into a signal. A two dimensional image is built as the light source moves along the retina.The interferometer integrates several data points over 2 mm depth to construct a tomogram of the structures.

TYPES AND DIFFERENCES

OCT may be time domain or spectral domain.

TIME DOMAIN OCT

1. In time domain OCT, the reference mirror moves back and forth. It moves one cycle for each axial scan so limits the speed of image acquisition.
2. Axial resolution is 10 microns, so takes 400 A scans/second.
3. Its a line scan, so there is limited amount of data and there is requirement for data interpolation.
4. Decentration effects occur.
5. Eye motion artifacts occur because of lesser axial resolution and scanning speed.

SPECTRAL DOMAIN OCT

1. In spectral domain OCT,the reference mirror is stationary and spectrometer analyses signal by wavelength.
2. Axial resolution is 5 microns and takes 20,000 A scans /second
3. Its a raster scan,so there is large amount of data and more precise segmentation of retinal layers
4. It has better repeatability and reliability.
5. There are no eye motion artifacts .

SCANS IN MIGRAINE

1.RNFL thickness report

Three concentric scans each of 1.34 mm radius centered around the ONH acquired in rapid succession(1.9 seconds scan time),each made of 256 A scans. It has been shown to exibit maximum reproducibility of RNFL measurement. The mean RNFL thickness is calculated using age adjusted RNFL thickness average analysis protocol. Third generation OCT offers a variety of RNFL thickness measurement and analysis protocols like RNFL thickness circle scan,fast circle scan, concentric three ring protocol, RNFL map and proportional circles

RNFL thickness is reported for clock hours, quadrants and as an overall average. RNFL thickness is usually superimposed on a normative database graph.In normal individuals RNFL thickness typically follows a “double hump” pattern. This is because RNFL is thicker at the inferior and superior poles of the nerve.

An individual patient's RNFL thickness profile can be compared to a normative database that represents the averaged RNFL profile from a large group of healthy subjects gathered at a variety of clinical sites. This profile is adjusted for age, because of natural loss of RNFL thickness with age.

In a normative database graph,

Green section encompasses **5th to 95th percentile** of normative range for RNFL.If the individual profile falls within the green area it is considered to be **within normal limits.**

Yellow section represents thickness for **first to fifth percentile** of the normal population range and is considered to be a "**borderline"** RNFL thickness.

Red section represents anything **less than the first percentile value** and is considered **"outside normal limits".**

Values greater than the 95th percentile cutoff are **above normal limits** and are indicated in **white**

Signal strength should be between 6 and 10.Thickness values from the three circumpapillary scans are averaged to generate the RNFL thickness profiles.

So the report consists of overall thickness value, averages by quadrants, averages by clock hours, table with various comparisons among maximum, minimum, and average RNFL thickness in quadrants and overall area.

RNFL thinning is noted in migraine.

OPTIC NERVE HEAD ANALYSIS REPORT

The “Fast Optic Disc scan” pattern consists of six evenly spaced radial lines centered on the ONH. Each of the six lines is made of 128 A scans crossing an area of 4.0 mm in length and the total scan time is 1.92 seconds.

The “Optic nerve head Analysis Report” printout shows both an individual B scan, which is an optical cross section that is selected by the technician. Overall grey scale, black and white fundus image are also shown in the report. Signal strength can range from 0 to 10.It provides an indicator of the quality of the image. Signal strength from 6 to 10 is generally considered acceptable.

The parameters in the “Individual Radial Scan Analysis” are primarily based on two automated algorithms: a segmentation algorithm that determines the location of the inner limiting membrane(blue line at the surface) and the one that detects the retinal pigment epithelium/Bruchs membrane termination(light blue circles with cross hairs inside). A second line in blue is drawn in parallel to the one connecting the two RPE/Bruch’s membrane termination points shifted up by the cup offset (typically 150 micrometers) to define the plane that separates the cup from the rim. This is shown by the red dotted line.

The cup diameter is the horizontal distance between the two intersections of the red dotted line with the ILM surface. Rim length is calculated as the Disc diameter minus the Cup diameter. The rim area for the vertical cross section parameter is the total area of the red shaded portion of the scan.The area is bordered by yellow vertical lines extending up from the RPE/Bruchs membrane termination,

the ILM surface, and the red dotted line separating cup and the rim. The average nerve head width at Disc is the thickness of the nerve fibres at the disc margin, indicated by the yellow lines extending up from the end of the RPE. These parameters are calculated individually for each of the six radial scans. However they provide only focal information along each individual radial line and are therefore of limited use by themselves.

The overall "Optic nerve head analysis results" is based on all the six radial scans, their segmentation and the interpolation of the data between each of the six radial scans. The six scans are aligned in a spoke pattern configuration and the smoothed lines are interpolated around the disc and cup margin points to define the disc(red) and cup(green).Disc and Cup area's are areas within these margins. Rim area is calculated as the Disc area minus the Cup area. Cup/Disc area ratio is calculated as a simple ratio of the areas. The Cup/Disc horizontal and Cup/Disc vertical ratios are calculated by taking the maximum cup diameter in the horizontal or vertical direction and dividing it by the maximum disc diameter in the horizontal or vertical direction. The Vertical integrated rim area is essentially the red shaded area in the individual scans interpolated around the disc to define a rim volume.The Horizontally integrated rim width takes the mean of of the average nerve widths and multiplies the value by the circumference of the disc.

The "Optic Nerve Head Analysis" and the" Individual radial scan analysis "reports along with RNFL thickness reports are very useful in assessing whether migraine is associated with other disorders like glaucoma.

TREATMENT OF MIGRAINE

Effective management of migraine headaches begins with making accurate diagnosis. Despite the fact that migraine is treatable ,many patients find themselves in a paradoxic situation in which they are both undertreated and overmedicated. They take or receive a large number of nonspecific analgesics which are minimally effective for migraine. It only contributes to analgesic rebound.

Treatment includes non pharmacologic as well as pharmacologic therapy.

Minimising excessive stress and sleep deprivation, eating regular meals, exercise and avoidance of common triggers (eg: wine, chocolate) are practical measures. Alternative therapies (biofeedback, relaxation, acupuncture) have been used successfully in conjunction with conventional medical treatment.

Headache diary should be maintained by every patient. It helps the physician and the patient characterize the headaches more clearly. It may also give the patients a sense of control over their illness.

ABORTIVE TREATMENT

1. **NSAIDS (nonsteroidal anti inflammatory drugs):**

They are appropriate agents for patients with mild to moderate migraine. Specific agents include acetaminophen, ibuprofen, naproxen sodium, and aspirin. Their effect is better when the drug is taken at the beginning of an attack, ideally during the prodromes.

2.TRIPTANS:

Triptans act on 5 HT 1B,1D receptors. They are serotonin receptor agonists..They produce vasoconstriction and reduce pain. They are useful for acute therapy of migraine. They are useful choices for patients with moderate to severe migraine. Intranasal and subcutaneous forms are available for patients who cannot tolerate oral drugs due to nausea. Triptans are contraindicated in patients with cardiovascular disease. Sumitriptan is widely used in treating migraine.

3.ERGOT ALKALOIDS:

Dihydroergotamine (DHE) and ergotamine are members of the ergot family used in migraine treatment.DHE is recommended for patients with moderate to severe migraine. DHE can be given subcutaneously, intravenously. intramuscularly or intranasally. Oral or rectal ergotamine may be useful for treating patients with migraine. They are generally taken at the beginning of an attack

4.OPIOID ANALGESICS:

Oral opioids may be useful when sedation will not place the patient at risk. Intranasal butorphanol, a mixed opiate receptor agonist-antagonist may be useful for some patients but it also carries the same risk of dependency.

5.ANTIEMETICS:

They are often used as adjuncts to other migraine therapies. Some drugs; eg: intravenous metoclopramide, intravenous and intramuscular prochlorperazine may be considered as monotherapy for migraine.Oral antiemetics are suggested only for adjunctive use.

PREVENTIVE TREATMENTS OF MIGRAINE

Preventive treatment is considered when the frequency of headache is so high that analgesic rebound is likely.It is also used when contraindications may limit the use and success of acute medications.

GROUP 1 DRUGS:

High proven clinical efficacy, mild to moderate adverse effects

Group 1 drugs include divalproex sodium (anticonvulsant), amitryptiline (tricyclic antidepressant), propranalol and timolol.

1.Divalproex sodium:

Class	:	anticonvulsants
Dose	:	500 – 1500 mg/day
Side effects	:	weight gain,tremor,alopecia,elevated liver enzymes
Contraindications	:	hepatic disease

Consider in patients with bipolar disorder, seizure disorder.

2.Propranolol

Class	:	beta blocker
Dose	:	80- 240 mg/day
Side effects	:	weight gain,syncope and depression

Contraindications : asthma, diabetes mellitus, depression

Consider in patients with cardiovascular disease, hypertension

3.Amitryptiline

Class : tricyclic anti-depressant

Dose : 50 -150 mg/day

Side effects : dry eyes and mouth,drowsiness

Avoided in patients with cardiac arrhythmias and elderly patients

Consider in patients with depression. tension type headache

GROUP 2 DRUGS

They have lower efficacy than group 1 drugs. They include atenelol (beta blocker), verapamil (calcium channel blocker), aspirin(NSAIDS), gabapentin (anticonvulsants), flouxetine (selective serotonin reuptake inhibitors, antidepressant), magnesium.

1.Gabapentin:

Class : anticonvulsant

Dose : 900 -2400 mg/day

Side effects : drowsiness,dizziness,weight gain

Contraindications : no specific contraindications

Considered in seizure disorder, neuropathic pain

2.Verapamil:

Class	:	calcium channel blocker
Dose	:	240 mg/day
Side effects	:	hypotension, constipation
Contraindications	:	orthostatic hypotension

Consider in patients with cardiovascular disease, migraine aura

GROUP 3 DRUGS

Group 3 drugs have efficacy based only on clinical experience, but no scientific evidence. This group includes nortryptiline and protriptiline (tricyclic anti-depressants), sertraline and paroxetine (selective serotonin reuptake inhibitors, antidepressants), topiramate (anticonvulsant).

GROUP 4 DRUGS

Group 4 drugs have medium to high efficacy but potentially significant side effects. They include flunarizine(anti emetic) and methysergide.

GROUP 5 DRUGS

Group 5 drugs have no evidence showing efficacy over placebo. This group includes clonidine (anti hypertensive), carbamazepine (anti convulsant) and indomethacin (NSAIDS).

OTHER DRUGS

Botulinum toxin is used recently for treating migraine. Treatment protocol generally involves injections of botulinum toxin into the head, facial and cervical musculature. The presumed mechanism involves decreasing or eliminating peripheral activation and thereby reducing central trigeminovascular activation.But till date there is no class A evidence favoring the routine use of botulinum toxin in patients with migraine.

OTHER TYPES OF HEADACHE

A.TRIGEMINAL CEPHALGIAS WITH AUTONOMIC FEATURES

Pain is usually present in the distribution of the ophthalmic nerve. Autonomic features often involving eyes are present. The headache usually affects the same side of the face and the head. Autonomic manifestations include eyelid edema, conjunctival injection, tearing, Horners syndrome, rhinorrhea,nasal congestion and facial flushing. Because of the prominent ocular manifestations these patients often seek ophthalmic care early in their course.

1.CLUSTER HEADACHE

It is one of the most painful headache disorders and is the most common trigeminal autonomic cephalgia.

INCIDENCE

Less common than migraine affecting less than 1% of the population

AGE OF ONSET

Between 27 and 31 years

SEX

The male to female ratio is 8:1

GENETICS

There seems to be a genetic component to cluster headaches in some families. It may affect monozygotic twins.However no specific gene is identified

MECHANISM AND PATHOPHYSIOLOGY

Both sympathetic(postganglionic Horners syndrome) and parasympathetic (lacrimation, rhinorrhea) nerves are affected. As in migraine there are both neuronal and vascular components.PET reveals bilateral activation in the region of the cavernous sinus, most prominent ipsilateral to headache, suggesting increased flow through cavernous portion of the carotid artery.MRI shows no abnormalities in the cavernous sinus area. The vascular changes may be due to trigeminal nerve activation as the observed features are not specific to cluster headache.

The parasympathetic features likely arise from superior salivatory nucleus.It innervates cerebral blood vessels as well as lacrimal and nasal mucosal glands accounting for the lacrimation,nasal congestion and rhinorrhea.

The trigeminovascular system involvement is evident by increased CGRP and VIP levels during attacks of cluster headache.

Suprachiasmatic nucleus also involved which regulates the rhythmic secretion of melatonin.

CLINICAL FEATURES

Pain quality	:	stabbing, boring
Pain severity	:	severe
Site	:	orbit and temple
Attacks per day	:	upto 8
Attack duration	:	15 – 180 minutes
Triggers	:	alcohol, nitroglycerine, histamine, REM sleep
Nocturnal attacks	:	present
Autonomic features	:	present

Photophobia and phonophobia are not as predominant as migraine.Nausea may occur but vomiting rarely occurs.In contrast to migraine,where the patients prefer to lie down in a dark quiet room,patients with cluster headache are restless and sometimes violent.They tend to pace,sit,rock,bang their head against the wall.The severity of the pain leads to suicidal thoughts

TYPES

1. Episodic-extends from 7 days to 1 year.

2. Chronic-extends for more than 1 year.

OPHTHALMIC FEATURES

Horners syndrome, lacrimation, conjunctival injection(autonomic features)

TREATMENT

Acute phase:

1. Inhaled oxygen within 15 min in 70% of patients.

2. Subcutaneous sumatriptan 6 mg produces headache relief within 15 minutes

3. IV dihydroergotamine

4. Sumatriptan and zolmitriptan nasal spray

Transitional prophylaxis:

The main aim is to shorten the cluster period and to obtain rapid remission. And also to reduce the headache frequency and severity.

1. Corticosteroids are used.Oral prednisolone (1 mg/kg body weight given only for three days) or dexamethasone (4 mg twice daily for 2 weeks ,then once daily for 1 week)

2. Ergotamine tartarate and dihydrotamine which is usually given for two or three weeks

Drugs for maintenance prophylaxis:

These drugs prevent recurrence during and after the transitional prophylaxis.

1. Verapamil starting at 240 mg daily and increasing to 720 mg daily in divided doses. It is well tolerated at very high doses. The common side effects are constipation, dizziness, peripheral edema, hypotension and bradycardia. Periodic ECG monitoring is advised

2. Lithium carbonate starting at 300 mg TDS or 450 mg sustained release. Common side effects are tremor, diarrhea and polyuria. Renal and thyroid functions should be monitored. Serum lithium concentrations and possible drug interactions should be monitored.

3. Valproic acid(250 mg BD upto 2 g daily)

4. Topiramate(50-125 mg daily)

5. Melatonin (10 mg)

6. Indomethacin is not much effective.

7. Methysergide was earlier used but now withdrawn due to pulmonary, retroperitoneal and pericardial fibrosis.

Surgery:

Considered for patients with exclusively unilateral headaches that cannot be controlled with standard medications.

1. Radiofrequency thermocoagulation of trigeminal ganglion is effective in 75% of patients with 20% recurrence rate. Complete analgesia must be produced,so long term corneal care is critical to prevent neurogenic ulceration.Other complications include diplopia, icepick pain and anaesthesia dolorosa.

2. Trigeminal sensory rhizotomy at the root exit zone.

3. Occiptal nerve stimulation

4. Deep brain stimulation of hypothalamus

PAROXYSMAL HEMICRANIA

Paroxysmal hemicranias may begin at any age but commonly starts in the fourth decade and is not familial. It may be chronic or episodic.

Pain quality	:	throbbing, boring, stabbing
Pain severity	:	severe
Site	:	orbit, temple
Attacks per day	:	1-40
Attack duration	:	2-25 min
Triggers	:	alcohol, bending head, pressing on neck or occipital nerve
Nocturnal attacks	:	present
Autonomic features	:	present
Female:male ratio	:	2:1

OPHTHALMIC FEATURES

1. Lid edema

2. Conjunctival injection

3. Horners syndrome

CHARACTERISTIC FEATURES

More common in women and are absolutely indomethacin positive

TREATMENT

1. Indomethacin is usually started at 25 mg tds and gradually increased to 50 mg tds.
2. Successful treatment with aspirin, verapamil, steroids and naproxen has been reported.

SHORT LASTING UNILATERAL NEURALGIFORM HEADACHES

This syndrome is the rarest of the trigeminal autonomic cephalgias. Headache is usually short lasting. It is present for 15-120 seconds, occurring upto 100 times daily. The pain is usually ocular. The pain starts and ends abruptly. Men are more commonly affected. Most patients have unilateral attacks and bilateral simultaneous attacks have also been reported.

Pain quality	:	burning,stabbing,electric
Pain severity	:	Moderate
Site	:	periorbital
Attacks per day	:	6-77
Attacks per day	:	5-250 seconds
Triggers	:	trigeminal trigger points (hair, forehead, face, nose, lip), facial and neck movements.
Nocturnal attacks	:	present

Autonomic features : present

Female : male ratio : 1:2

Age at onset is 23 to 77 years. Symptomatic periods may last from days to months.

OPHTHALMIC FEATURES

1. Conjunctival congestion
2. Ptosis
3. Lacrimation
4. Lid edema

Intaocular pressure and systolic blood pressure may be increased during the attacks. Intraorbital tumors, pituitary tumors, brainstem tumors and vascular malformations have been found in some patients. So neuroimaging is recommended for all patients.

TREATMENT

Treatment is largely ineffective. Most migraine preventive medications have been tried. Some success has been reported using lamotrigine, topiramate, carbamazepine, verapamil,valproate and nifedipine.

SHORT LASTING HEADCHES(UNILATERAL AND NEURALGIFORM)

The condition is extremely rare. It consists of severe, sharp, unilateral retroorbital headaches lasting from 40 to 80 seconds but occasionally upto 30 minutes. Attacks occur 20 – 30 times daily without trigger points. There is a profuse unilateral tearing without other autonomic features.

Pain quality	:	sharp
Pain severity	:	severe
Site	:	retroorbital
Attacks per day	:	20-30
Attack duration	:	40-80 seconds(rarely upto 30 minutes)
Triggers	:	none
Nocturnal attacks	:	absent
Female:male ratio	:	unknown

OPHTHALMIC FEATURES

Lacrimation

TREATMENT

Gabapentin is minimally effective

PRIMARY HEADACHE DISORDERS CAUSING OCULAR OR PERIOCULAR PAIN

PRIMARY STABBING HEADACHE

This type of headache is also known as icepick pains, jabs and jolts, ophthalmodynia periodica. It affects 1 -2% of the population. It occurs more frequently in patients with cluster headache, TTH, cervicogenic headache, hemicrania continua. Primary stabbing headache may start at any age and primarily affects women.

The attacks are very short, lasting a second and continues upto 10 seconds. The pain frequently occurs in or near the eye but tends to migrate. There are generally no provoking factors. The condition is generally benign.

If the attacks are frequent enough to warrant prophylactic therapy, indomethacin, melatonin,celecoxib and amitryptilline are useful.

HEMICRANIA CONTINUA

Hemicrania continua is a continuous, unilateral headache that fluctuates in intensity. Young women are more commonly affected. Pain is generally located in the anterior head.It is usually dull aching or pressing type of pain. Some patients have autonomic symptoms such as conjunctival injection, lacrimation, nasal congestion. rhinorrhea, ptosis, lid edema. Photophobia, phonophobia, nausea and vomiting are common.There may be superimposed primary stabbing headaches. Patients are indomethacin responsive.

INDICATIONS FOR NEUROIMAGING IN PATIENTS WITH HEADACHE

1. The first or worst headache of patients life, particularly abrupt onset
2. Change in frequency, severity or clinical features of migraine attack
3. Neurological symptoms that donot meet criteria for migraine with aura
4. Hemicrania that is always on the same side
5. Positional headache
6. No improvement with conventional therapy

PART 2

AIMS AND OBJECTIVES

To elucidate the various ophthalmic manifestations of different types of migraine

PRIMARY OBJECTIVES

1. To elucidate the various ophthalmic manifestations of different types of migraine
2. To assess the response to treatment(with steroids) in Ophthalmoplegic migraine.
3. To assess the visual field changes in migraine.
4. OCT changes in migraine.

SECONDARY OBJECTIVES

1. Association with other ocular disorders

STUDY DESIGN

Prospective, clinical study

PATIENT SELECTION

All migraine cases with typical symptoms and signs in the period of 1 year by the method of random sampling.Age of inclusion was 5 to 60 years. Both genders were selected.

INCLUSION CRITERIA

1. Patients with migraine (migraine with aura and migraine without aura).
2. Retinal migraine
3. Ophthalmoplegic migraine

EXCLUSION CRITERIA

1. Any other type of headache other than migraine
2. Refractive errors.
3. Head or eye trauma
4. Eyes with retinal or optic disc pathology
5. Cataract
6. Corneal opacity
7. Ocular laser treatment
8. CNS disorders like brain tomours,infarction,encephalitis,parkinsons
9. Diabetic and hypertensive retinopathy

SAMPLE SIZE

50 patients

AGE

5 to 60 yrs

STUDY PARAMETERS

1. Detailed history of present and past illness
2. Drug and treatment history
3. Visual acuity using Snellen's acuity chart
4. Refraction
5. Colour vision
6. Slit lamp biomicroscopy of anterior segment
7. Intraocular pressure using Goldmann Applanation tonometry
8. Direct and indirect ophthalmoscopy
9. Gonioscopy
10. Automated perimetry
11. Optical coherence tomography

DATA COLLECTION AND METHODOLOGY

50 patients based on the inclusion criteria were selected and evaluated subsequently. Various clinical findings of migraine were elucidated. All the above investigations were done. Follow up done at 1,4,6 weeks and late follow up at 3,6 and 12 months

METHODS

All patients included in the study were evaluated first by measuring the visual acuity using Snellens visual acuity chart at 6 meter distance. Detailed history was taken. History of headache (type, areas involved, association with vision loss, associations with aura, nausea, vomiting, field loss) and history of associated visual phenomena of migraine (scintillation scotoma, fortification spectra, micropsia, metamorphopsia, macropsia, pallinopsia, cerebral polyopia, Alice in wonderland syndrome, achromatopsia) were noted. History of visual snow and photophobia also noted. Past history of ophthalmic (glaucoma and retinal disease) and systemic diseases (diabetes, hypertension, epilepsy, cardiovascular disease, renal failure and CNS disorders).Treatment history noted(treatment for migraine or any other systemic diseases and the duration of treatment).

Refraction was done for all patients. Anterior segment evaluation was done using slitlamp (lid edema, conjunctival congestion, Horners syndrome common in other types of headache other than migraine like cluster headache, paroxysmal hemicranias. short lasting unilateral neuralgiform headaches).

Colour vision was checked for the patients in the study. Dilated fundus examination was done to rule out other retinal disorders like diabetic or hypertensive retinopathy, retinal detachment, macular disorders).

Intraocular pressure was checked using Goldmann's applanation tonometry. Angle study was done using gonioscopy. Fields evaluated using standardized automated perimetry (Octopus).Optical coherence tomography was also done for all patients to see if there are any optic disc changes and retinal nerve fibre layer

thinning. These tests were helpful to find out if migraine is associated with any other ocular disorders like glaucoma.

In ophthalmoplegic migraine , extraocular movements were checked and diplopia charting was done as it was associated with nerve palsies.Visual acuity, refraction, fields (using automated perimetry), colour vision, fundus (direct and indirect ophthalmoscopy) were checked. Patients were started with steroids along with anti migraine medications and its response to treatment was studied.

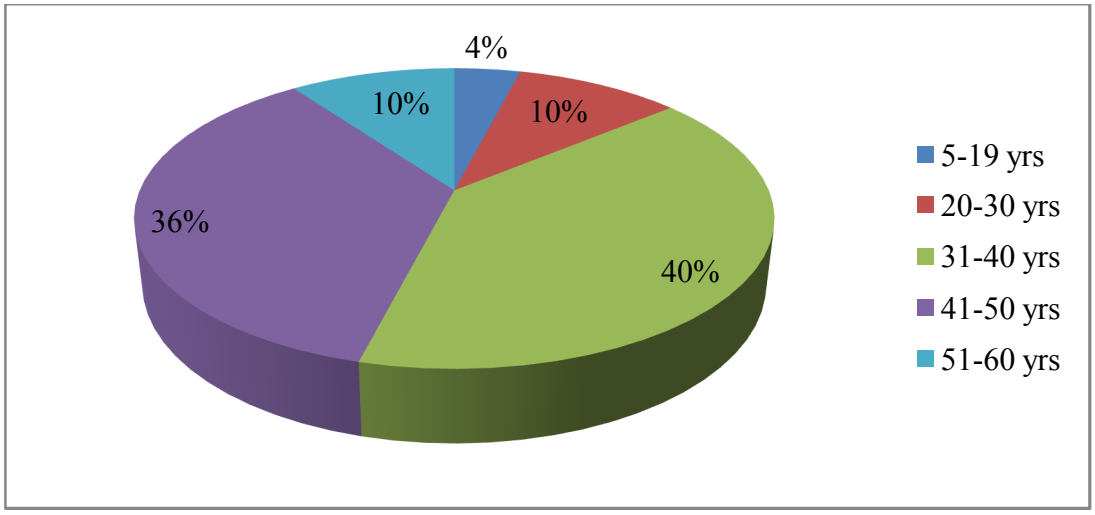
OBSERVATION AND RESULTS

AGE

Table 1:Age distribution of the study patients

AGE IN YEARS	NUMBER OF PATIENTS AND PERCENTAGE
5-19	2(4 %)
20-30	5(10 %)
31-40	20(40 %)
41-50	18(36 %)
51-60	5(10 %)

Figure 1: Age distribution of study patients



Out of the 50 patients examined,2 patients:5 years old (ophthalmoplegic migraine), 5 patients:20-30 years of age,20 patients:30-40 years of age,18 patients:40-50 years of age

5 patients:50-60 years of age. The mean age of presentation was around 40.58 years of age.

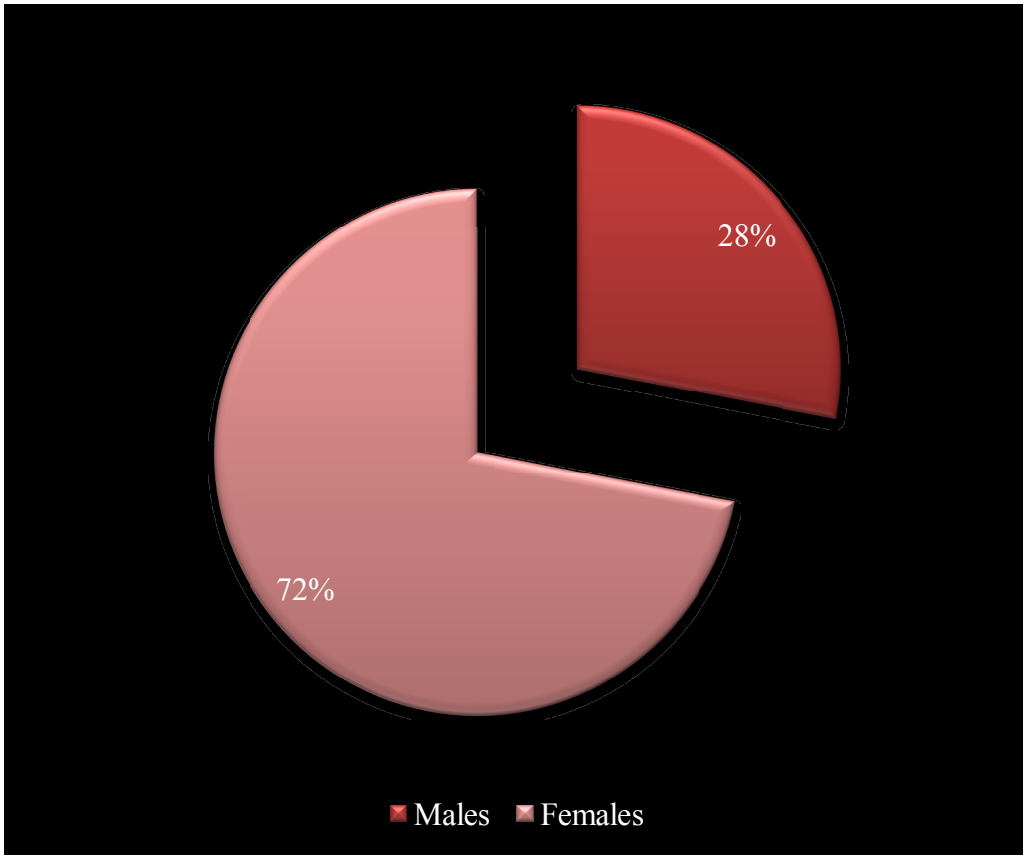
SEX

Out of the 50 patients examined,36 patients were females(including the paediatric age group) and 14 patients were males.

Table 2:Sex distribution of the patients

SEX	NUMBER OF PATIENTS AND PERCENTAGE
Males	14(28 %)
Females	36(72 %)

Figure 2: Sex distribution of the patients



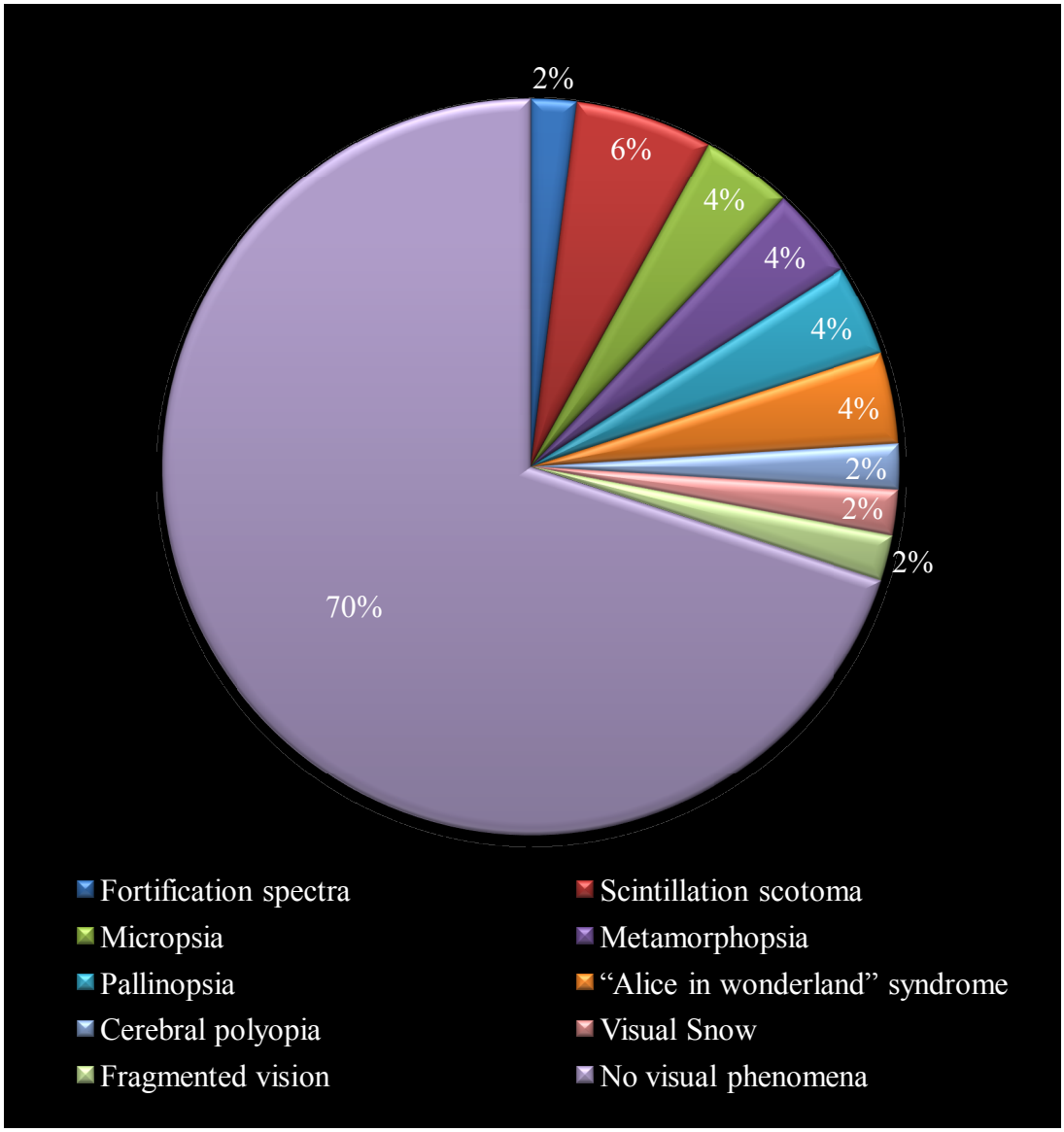
VISUAL PHENOMENA

All the patients were asked about the visual phenomena experienced by them during the attack of migraine. History of scintillation scotoma, fortification spectra, micropsia, macropsia, matamorphopsia, pallinopsia, cerebral polyopia, Alice in wonderland syndrome, achromatopsia were asked.

Table 3:Visual phenomena of the patients in the study

VISUAL PHENOMENA	NUMBER OF PATIENTS
Fortification spectra	1(2%)
Scintillation scotoma	3(6%)
Micropsia	2(4%)
Macropsia	0
Metamorphopsia	2(4%)
Pallinopsia	2(4%)
“Alice in wonderland” syndrome	2(4%)
Cerebral polyopia	1(2%)
Achromatopsia	0
Visual Snow	1(2%)
Kaleidoscopic vision	0
Fragmented vision	1(2%)
Mosaic vision	0

Figure 3:Visual phenomena in the study patients



Out of the 50 patients examined 15 patients (30%) had some visual phenomena associated with migraine attacks.

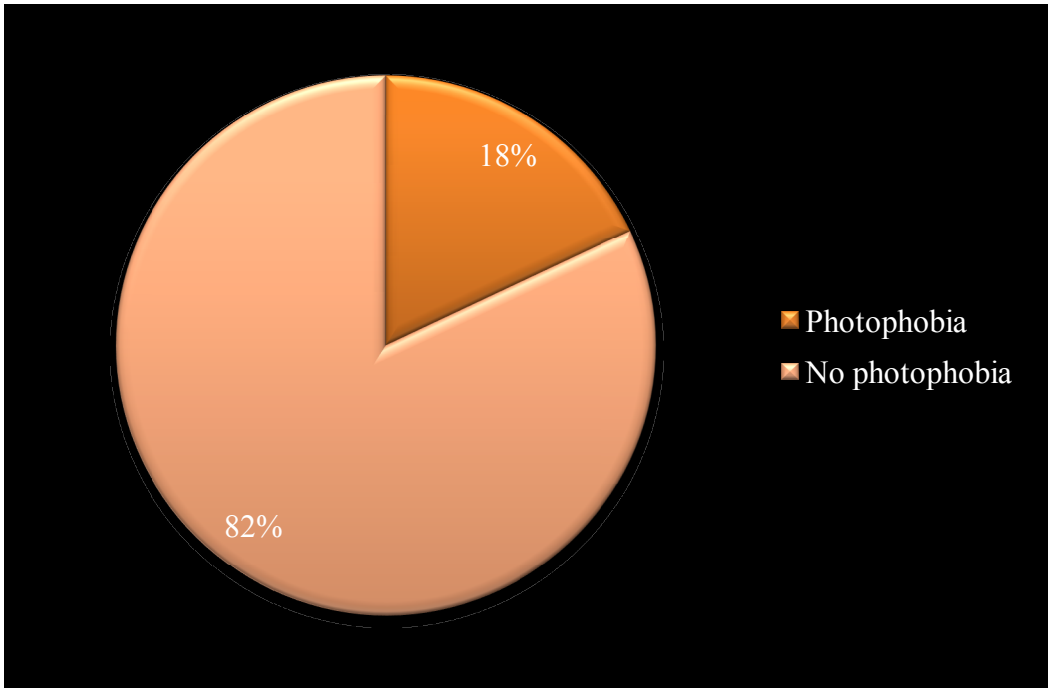
OTHER ASSOCIATED VISUAL FEATURES

Out of the 50 patients examined 9 patients had photophobia during a migraine attack. No patients had persistent visual field defects like homonymous hemianopia or tunnel vision or persistent loss of vision(negative symptoms.

Table 4:Associated visual features in the study patients

ASSOCIATED VISUAL FEATURES	NUMBER OF PATIENTS AND PERCENTAGE
Photophobia	9(18%)
Negative symptoms	0

Figure 4:Associated visual features in the study patients



COLOUR VISION:

All 50 patients examined had normal colour vision.

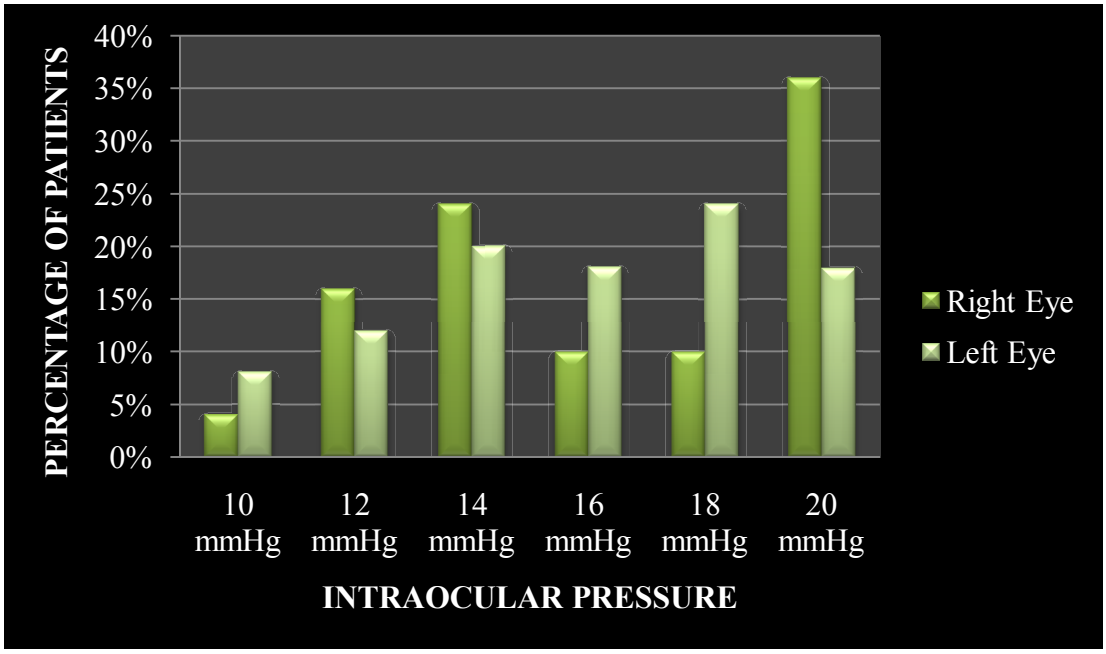
INTRAOCULAR PRESSURE

Intraocular pressure was measured for all 50 patients by Goldmann's applanation tonometry.IOP was within normal limits for all patients for both the eyes

Table 5:Intraocular pressure of the study patients

RE	LE
10-15 mmHg: 22 patients	10-15 mmHg:20 patients
16-20 mmHg: 28 patients	16-20 mmHg:30 patients
10 mmHg: 2(4%)	10 mmHg: 4(8%)
12 mmHg: 8(16%)	12 mmHg: 6(12%)
14 mmHg: 12(24%)	14 mmHg: 10(20%)
16 mmHg: 5(10%)	16 mmHg: 9(18%)
18 mmHg: 5(10%)	18 mmHg: 12(24%)
20 mmHg: 18(36%)	20 mmHg: 9(18%)

Figure 5:Intraocular pressure of the study patients



REFRACTION

Retinoscopic refraction was done for all patients.Presbyopic correction was needed for 25 patients

>35 years of age:2 patients

>40 years of age:23 patients(18 patients:40-50 years of age,5 patients:50-60 years of age)

FUNDUS EXAMINATION

All patients in the study were subjected to dilated detailed fundus examination by direct, indirect ophthalmoscopy and slit lamp biomicroscopy using 90D lens.

BE Fundus was found to be normal for all patients.BE Peripheries was also found to be normal for all patients.

GONIOSCOPY

Shaffers grading of angles was done for all the 50 patients. Goldmann's single mirror lens was used to grade the angles. All patients had open angles in both eyes.

AUTOMATED PERIMETRY

All patients in the study were subjected to examination of visual fields using standardized automated perimetry (Octopus).All patients had normal visual fields in both eyes.

OPTICAL COHERENCE TOMOGRAPHY

All patients underwent optic nerve head analysis and RNFL thickness testing

RNFL THICKNESS REPORTS

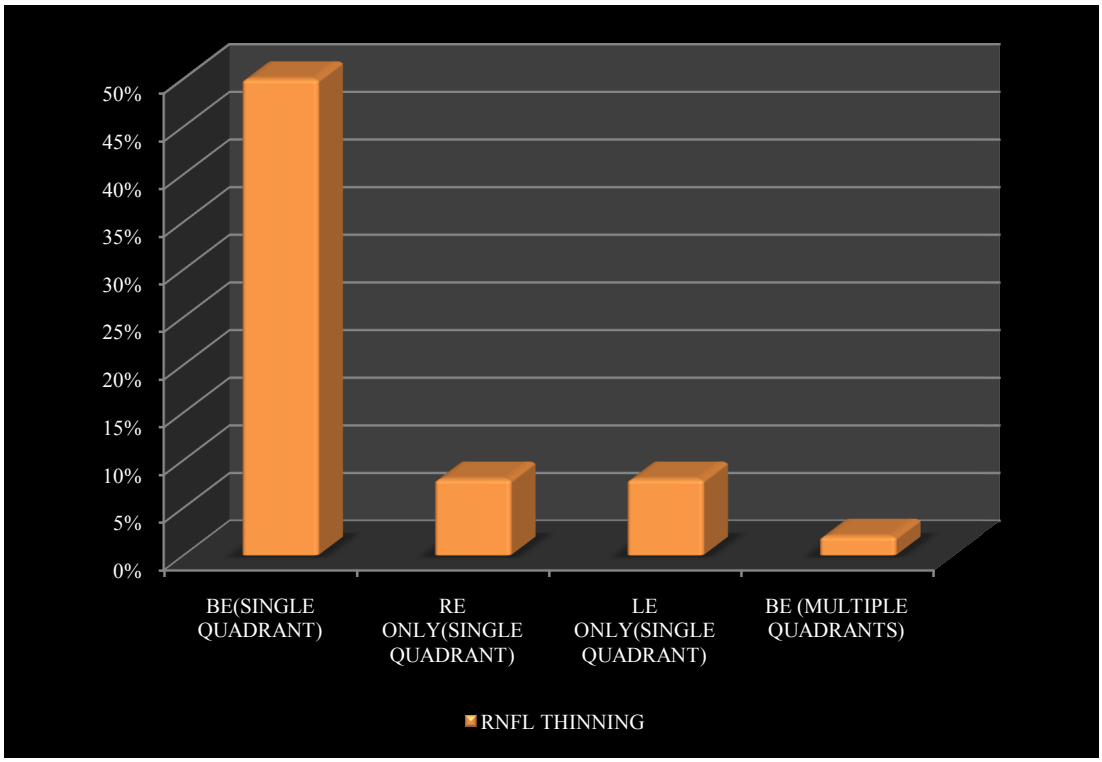
BE RNFL thinning noted in 25 patients(50%) (single quadrant thinning)

RE RNFL thinning alone noted in 4 patients(8%) (single quadrant thinning)

LE RNFL thinning alone noted in 4 patients(8%) (single quadrant thinning)

BE more than 2 quadrants thinning noted in one patient(2%)

Figure 6: RNFL thickness reports in the study patients



RE:

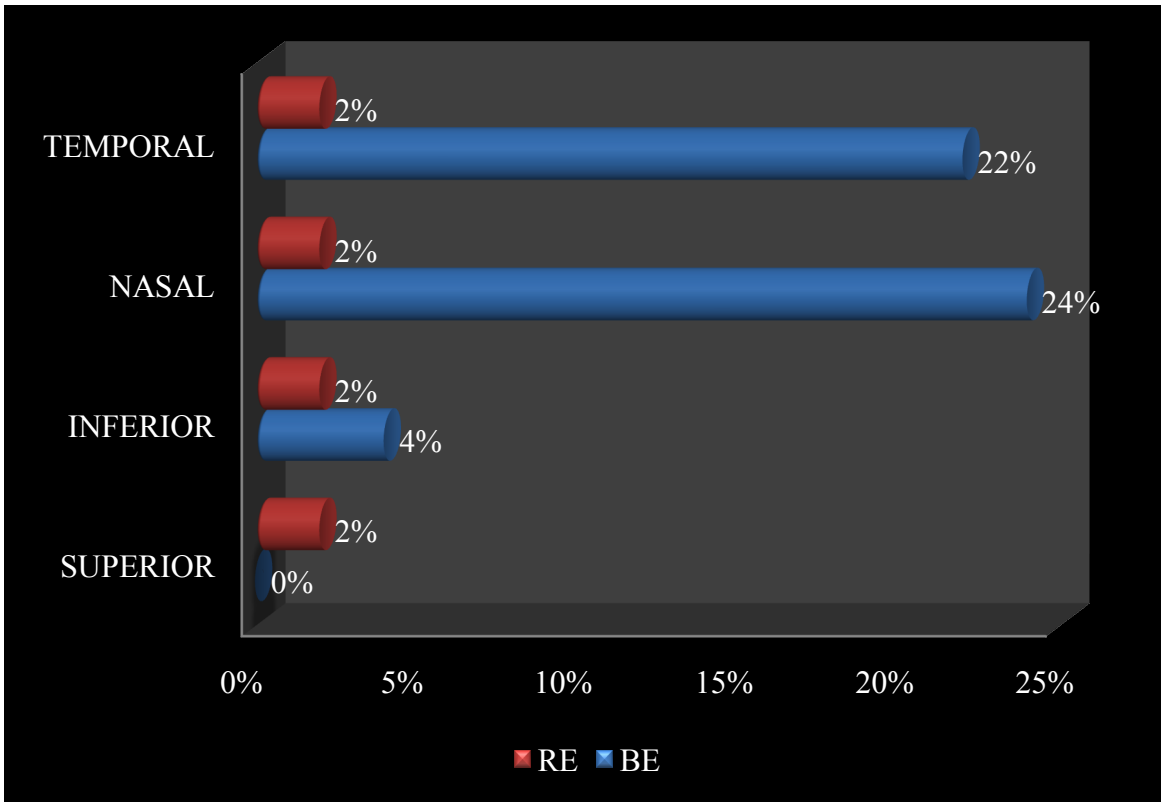
Nasal quadrant thinning alone noted in 13 patients(26%).Out of which 12 patients(24%) had BE thinning and 1 patient(2%) had RE thinning alone.

Temporal quadrant thinning noted in 12 patients(24%).Out of which 11 patients(22%) had BE thinning and 1 patient(2%) had RE thinning alone.

Superior quadrant thinning noted in 1 patient(2%) with RE thinning alone. Inferior quadrant thinning noted in 1 patient(2%)with RE thinning alone.

Inferior quadrant thinning noted in 2 patients(4%) with BE thinning(in RE)

Figure 7:RNFL thinning in RE of the study patients(RE alone & RE thinning in BE involvement.The other eye involvement may be any quadrant)



LE:

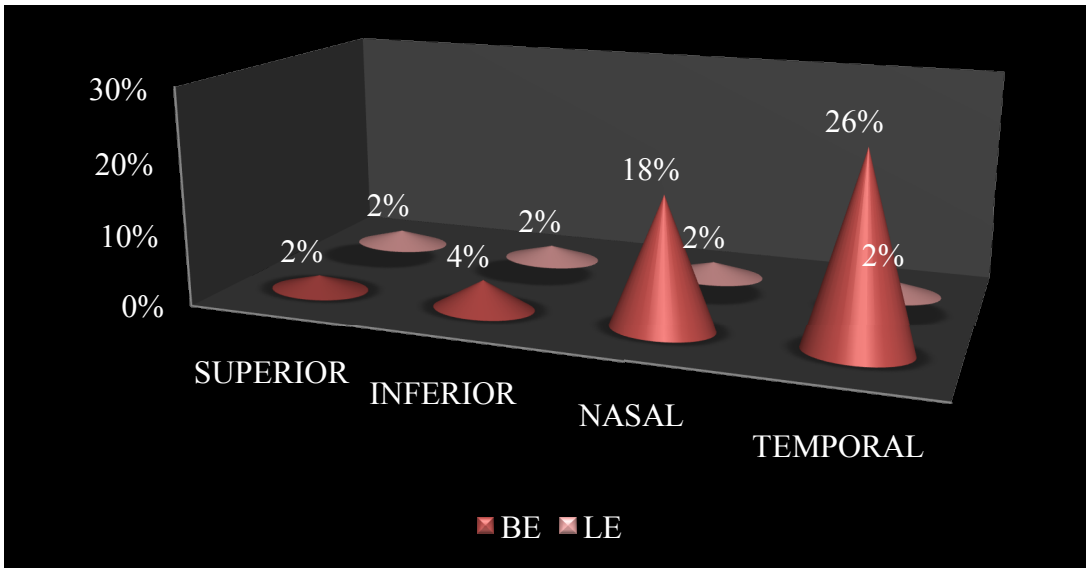
Nasal quadrant thinning alone noted in 10 patients(20%).Out of which 9 patients(18%) had BE thinning and 1 patient(2%) had LE nasal quadrant thinning alone

Temporal quadrant thinning noted in 14 patients(28%).Out of which 13 patients(26%) had BE single quadrant thinning and 1 patient(2%) had LE temporal quadrant thinning alone.

Inferior quadrant thinning noted in 3 patients(6%).Out of which 2 patients(4%) had BE single quadrant thinning and one patient(2%) had LE inferior quadrant thinning alone

Superior quadrant thinning noted in 2 patients(4%).Out of which 1 patient(2%) had BE single quadrant thinning and 1 patient(2%) had LE superior quadrant thinning alone.

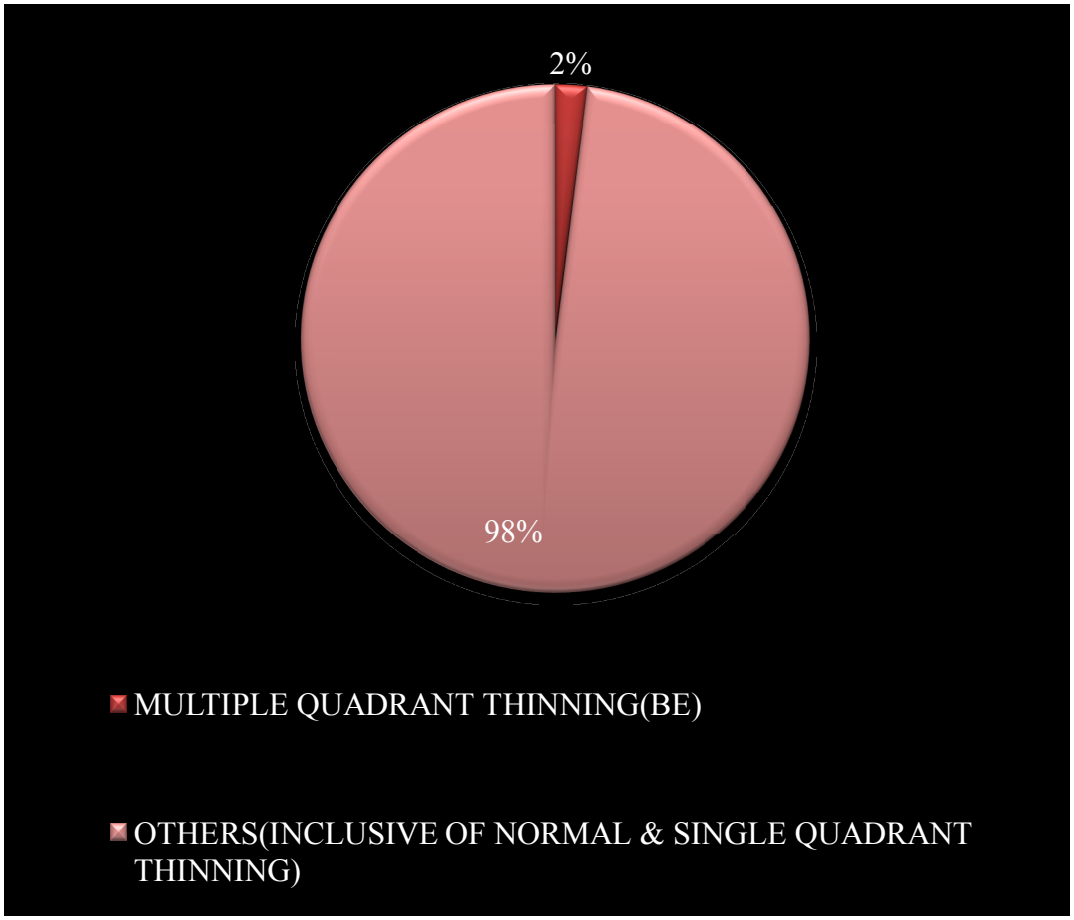
Figure 8:RNFL thinning in LE of the study patients(LE alone & LE involvement in BE thinning.The other eye involvement may be any quadrant)



MULTIPLE QUADRANT THINNING

Out of the 50 patients ,1 patient had superior,nasal and temporal quadrant thinning in RE and nasal ,temporal quadrant thinning in LE.

Figure 9:Multiple quadrant thinning(BE) in the study patients



OPHTHALMOPLEGIC MIGRAINE

Out of the 50 patients examined 2 patients had ophthalmoplegic migraine.Both the patients were young female children of 5 years of age.

HISTORY

One child was referred as a case of ophthalmoplegic migraine for ophthalmic evaluation.Another child attended our outpatient department with a classical history.Both the children presented with the following history

Child 1:

1. History of drooping of RE upper lid for 1 week duration, with no history of change in drooping while facial movements like chewing. No history of fluctuations and fatiguability.

2. History of headache for 1 week duration. It was throbbing in nature, more in the right frontal region with history of intolerance to the noisy environment and head banging over walls (temper tantrums)

3. History of defective vision RE for 1 week duration mainly for distance

Child 2:

1. History of drooping of LE upper lid for 5 days duration, with no history of change in drooping while facial movements like chewing.No history of fluctuations and fatiguability.

2. History of headache for 5 days duration. It was throbbing in nature, more in the left frontal region with history of intolerance to the noisy environment.

3. History of defective vision LE for the past 5 days mainly for distance

Both the children had no history of seizures, loss of consciousness, head injury, neck stiffness. No history suggestive of 1,2,4,5,6,7,9,11,12 th cranial nerves involvement. There was no history of weakness of upper and lower limbs, difficulty in speech or swallowing or walking. There was no history of reduced sensations over any part of the body. There was no history of fever or trauma or loose stools.

PAST HISTORY

First child had recurrent attacks of right sided headache on and off for the past one and half years and was diagnosed as a case of migraine. She was treated with oral propranalol. Patient was asked to continue the medications but defaulted. There was no history of sinusitis or systemic illness like tuberculosis.

Second child was diagnosed as a case of migraine and she was on regular medications(T. Propranalol 40 mg ½ TDS and T.Amitryptilline 10 mg ¼ HS).

Antenatal, natal, postnatal, developmental, immunization history were normal for both the children

FAMILY HISTORY

The mother of the first child had migraine since she was 15 years of age.But she was on irregular treatment

ANTERIOR SEGMENT EXAMINATION

Child 1:

Facial asymmetry was present.

Head posture:normal

Right eye		**Left eye**
6/24 NIP	Vision	6/9 NIP
All movements except abduction and intorsion restricted	Extraocular movements	Full
Clear	Conjunctiva	Clear
Clear	Cornea	Clear
Normal depth	Anterior chamber	Normal depth
Normal colour pattern	Iris	Normal colour pattern
Sluggishly reacting to light,4 mm	Pupil	3 mm reacting to direct and consensual light reflexes
Clear	Lens	Clear
Normal	Fundus	Normal
Normal	Colour vision	Normal

Child 2

Facial asymmetry was present.

Head posture was normal

RIGHT EYE		**LEFT EYE**
6/6	Vision	6/12 NIP
Full	Extraocular movements	**All movements except intorsion and abduction restricted**
Clear	Conjunctiva	Clear
Clear	Cornea	Clear
Normal depth	Anterior chamber	Normal depth
Normal colour pattern	Iris	Normal colour pattern
3 mm reacting to both direct and consensual light reflexes	Pupil	**4mm,sluggishly reacting to light**
Clear	Lens	Clear
Normal	Fundus	Normal
Normal	Colour vision	Normal

ORTHOPTIC EVALUATION

Child 1:

1. Done after retracting RE upper lid.
2. Cover test : RE divergent squint with hypotropia
3. Extraocular movements: All movements except abduction and intorsion restricted
4. Prism bar cover test : FR Exotropia > 90 prism dioptres, FL 30 prism dioptres.
5. Secondary deviation more than primary deviation.

Child 2:

1. Done after retracting LE upper lid.
2. Cover test:LE divergent squint with hypotropia
3. Extraocular movements : All movements except abduction and intorsion restricted.
4. Prism bar cover test : FR exotropia 30 prism dioptres, FL 90 prism dioptres
5. Secondary deviation more than primary deviation

PTOSIS EVALUATION:

Both the children had complete ptosis (first child had RE ptosis and second child had LE ptosis). Lid crease was present in both the children. Ice pack and fatiguability tests were negative in both the children. There was no lagophthalmos and bells phenomenon was intact in both the children.

DIPLOPIA CHARTING

Both the children had crossed diplopia on opening the ptotic lid.

NEUROLOGIST OPINION:

Both the children were advised MRI with contrast.CSF analysis, thyroid profile and vasculitis work up

INVESTIGATIONS TAKEN:

1. BASELINE

CHILD 1	**INVESTIGATIONS**	**CHILD 2**
11.6 g/dl	Hemoglobin	11 g/dl
10,800 cells/cubic mm	Total count	10,200 cells/cubic mm
P 48% L 52%	Differential count	P 48% L 52%
3,75,000/cubic mm	Platelet count	3,50,000/cubic mm
33.3%	Packed cell volume	36%
22 mg/dl	Blood urea	25 mg/dl
0.5 mg/dl	Serum creatinine	0.4 mg/dl
138 mEq/L	Serum sodium	140 mEq/L
4.2 mEq/L	Serum potassium	3.9 mEq/L
28 IU/ml	SGOT	36 IU/L
29 IU/ml	SGPT	32 IU/L
Normal	Chest X ray	Normal

2.VASCULITIS WORKUP

CHILD 1		CHILD 2
6 mm/16 mm	ESR	7 mm/12 mm
Negative	CRP	Negative
Negative	ANA	Negative
Negative	ANCA	Negative
Negative	RF	Negative

3.THYROID PROFILE

CHILD 1		CHILD 2
99.56 ng/dl	Serum T3	110 ng/dl
8.02 micrograms/dl	Serum T4	6.5 micrograms/dl
2.670 mIU/ml	Serum TSH	3.25 mIU/ml

4.NEUROIMAGING

CHILD 1	NEUROIMAGING	CHILD 2
Normal	CT brain with angiography	Normal
Normal	MRI brain	Normal
Normal	MRA & MRV	Normal

5.CSF ANALYSIS

CHILD 1	CSF ANALYSIS	CHILD 2
56 mmol/L	Glucose	58 mmol/L
90 g/L	Protein	85 g/L
No growth	Culture & sensitivity	No growth
P 0% L 1%E 0%	Cell count	P 0% L 1% E 0%
Negative	Latex	Negative

6.METABOLIC PARAMETERS

CHILD 1	METABOLIC PARAMETERS	CHILD 2
0.52 mmol/L	Serum lactate	0.48 mmol/L
9.0 micromol/L	Serum ammonia	8.4 micromol/L
50 mg	Urine porphyrin	52 mg

7.OTHERS

CHILD 1		CHILD 2
C3=96.1 mg/dl, C4=18.5 mg/dl	Complement factors	C3=92.5 mg/dl, C4=18.5 mg/dl
Normal	Echocardiogram	Normal

TREATMENT GIVEN

Both the children were treated with

1. T.Propranalol 40 mg ½ TDS
2. T.Amitryptilline 10 mg HS
3. T.Prednisolone 20 mg was started and continued for 15 days and then gradually tapered for the next 1 month.
4. T.Ranitidine 150 mg ½ BD

FOLLOW UP

Both the children were followed up for the next 3 months

After 1 month:

Child 1: RE ptosis recovered completely. Movements in RE recovered partially.RE pupil was reacting to both direct and consensual light reflexes.There was residual exo 45 prism dioptres in the RE.

Child 2: LE ptosis recovered completely. Movements in LE recovered partially. LE pupil was reacting to both direct and consensual light reflexes. There was residual exo 30 prism dioptres in the LE.

After 2 months:

Child 1 & 2

Vision(BE):6/6

Colour vision(BE):Normal

EOM:Full

Diplopia charting:No diplopia

Fundus(BE):Media clear

Disc and vessels:normal

Macula:FR present

There was residual exo of 15 prism dioptres RE for the first child and 10 prism dioptres in the LE for the second child

A CASE OF OPHTHALMOPLEGIC MIGRAINE

Figure 10 a

Figure 10 b

Figure 10 a: 5 yr old child with RE ophthalmoplegic migraine(ptosis was present)

Figure 10 b: Restriction of adduction in RE,pupil:4 mm sluggishly reacting to light.Vision(RE):6/24 NIP;(LE):6/9 NIP

Figure 10 c

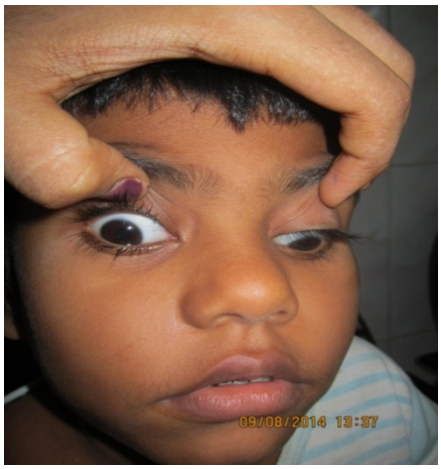


Figure 10 d

Figure 10 c: Restriction of elevation in RE

Figure 10 d: Restriction of depression in RE

A CASE OF OPHTHALMOPLEGIC MIGRAINE

Figure 10 e: Abduction was full in RE

FOLLOW UP AFTER 1 MONTH(PATIENT STARTED ON STEROIDS)

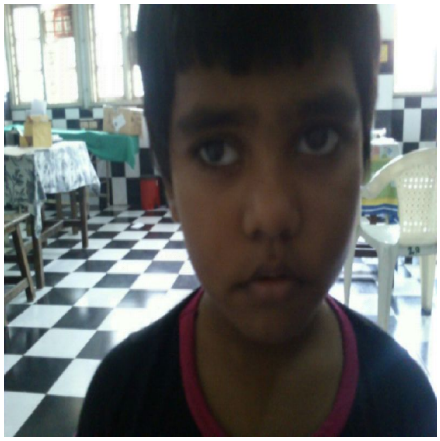


Figure 11: Ptosis recovered fully.Extraocular movements recovered partially.There was residual exo of 45 prism dioptres in RE.Pupil was reacting to both direct and consensual light reflexes.

FOLLOW UP AFTER 2 MONTHS

Figure 12 a: The child had residual exo of 15 prism dioptres in RE,with vision 6/6,BE pupils reacting to light reflexes

Figure 12 b: Abduction and adduction were full

Figure 12 c:Elevation and depression were full

DISCUSSION

The study was conducted in a tertiary care hospital and 50 patients based upon the inclusion criteria were selected.

Out of the 50 patients,2 patients belonged to paediatric age group(5 years of age).They were diagnosed as ophthalmoplegic migraine after all the necessary investigations and treatment was given. The mean age of presentation was 5 years of age. All the patients were subjected to a list of investigations.

Out of the 50 patients,10% of patients belonged to 20-30 years of age.40% of patients belonged to 31-40 years of age.36% of patients belonged to 41-50 years of age.10% of patients belong to 51-60 years of age.The mean age of presentation was around 40.58 years of age.

Out of the 50 patients,28% of patients were males and 72% of patients were females. So according to the study, females were more commonly affected than males.

Out of the 50 patients examined,2% of patients had fortification spectra.6% of patients had scintillation scotoma.4% had micropsia.4% of patients had metamorphopsia.4% of patients had pallinopsia.4% of patients had “Alice in wonderland”syndrome.2% of patients had cerebral polyopia.2% of patients had visual snow.2% of patients had fragmented vision.

Totally around 30% of patients had visual phenomena associated with migraine attacks.

Around 18% of patients had photophobia.

All patients had normal colour vision, normal range of intraocular pressure.

Gonioscopy showed open angles for all patients.

Fundus was found to be normal in all patients. Peripheries were found to be normal in all patients.

Refraction was done in all patients.Presbyopic correction was given to 25 patients.

Automated perimetry was found to be normal in all patients.

Optical coherence tomography was done in all patients. BE RNFL thinning (single quadrant thinning)was found in 50% of patients.RE RNFL thinning (single quadrant thinning)was found in 8% of patients. LE RNFL thinning(single quadrant thinning) was found in 8% of patients. BE more than 2 quadrants thinning noted in 2% of patients

In the RE, nasal quadrant thinning alone is noted in 26% of patients. Out of which 24% of patients had BE thinning and 2% had RE thinning alone. Temporal quadrant thinning noted in 24% of patients. Out of which 22% of patients had BE thinning and 2% of patients had RE thinning alone. Superior quadrant thinning noted in 2% of patients with RE thinning alone. Inferior quadrant thinning noted in 2% of patients with RE thinning alone. Inferior quadrant thinning noted in 4% of patients with BE thinning(in RE)

In LE, nasal quadrant thinning alone noted in 20% of patients. Out of which 18% of patients had BE RNFL thinning and 2% of patients had LE nasal quadrant thinning alone. Temporal quadrant thinning noted in 28% of patients. Out of which 26% of patients had BE single quadrant thinning and 2% of patients had LE temporal quadrant thinning alone. Inferior quadrant thinning noted in 6% of patients. Out of which 4% of patients had BE single quadrant thinning and 2% of patients LE inferior quadrant thinning alone. Superior quadrant thinning noted in 4% of patients. Out of which 2% of patients had BE single quadrant thinning and 2% of patients had LE superior quadrant thinning alone.

Out of the 50 patients,2% of patients had multiple quadrant thinning.

Out of the 50 patients examined,2 patients had ophthalmoplegic migraine. Both the children were young female children of 5 years of age. Both the children presented with history of throbbing headache ,drooping of upper eyelid (RE for the first child and LE for the second child) of acute onset and short duration. All extraocular movements except intorsion and abduction were restricted(RE in first child and LE in second child).Both the children had ptosis (RE for the first child and LE for the second child).Both the children had past history of migraine. Incomitant squint (RE for the first child and LE for the second child) was present in both the children. Both the children had crossed diplopia on opening the ptotic lid. Both the children were subjected to baseline, metabolic, thyroid profiles, CSF analysis, neuroimaging. All investigations found to be normal. Neurologist and cardiologist opinions were obtained. Both the children were started with T.Prednisolone 20 mg 1 OD, continued for 15 days and tapered gradually for the next 1 month. They were advised to continue T.Propranalol 40 mg ½ TDS, T.Amitryptilline 10 mg HS. Both the children gradually improved and became normal after 2 months(BE vision:6/6,ptosis improved completely, EOM:full, no diplopia)

CONCLUSION

The visual phenomena which occurs in migraine is due to the cortical spreading depression. Aura symptoms are produced in relation to the areas of cerebral cortex affected. Primary and visual association cortex is associated with visual symptoms of migraine.

The mean age of presentation of migraine was 40.58 years of age in the study. It was more common in females than males. Visual phenomena associated with migraine was present in 30% of patients with migraine. Photophobia was present in 18% of patients. Colour vision, fundus, automated perimetry, slit lamp examination, gonioscopy were found to be normal in all patients. RNFL thinning was noted by OCT in BE- 50% of patients(single quadrant thinning), RE:8% of patients (single quadrant thinning),LE:8% of the patients(single quadrant thinning),multiple quadrant thinning:2% of patients.

All patients were already on treatment with T.Propranalol 40 mg(either ½ BD or 1 BD), T.Amitryptilline 25 mg(either ½ HS or 1 HS),T.Para 500 mg s-o-s as directed by the neurologist. Patients who experienced visual phenomena did so when they missed medications (more when they were on irregular treatment)

Patients with ophthalmoplegic migraine responded to treatment with steroids and completely recovered in 2 months. The mean age of presentation was 5 years of age. These patients belonged to paediatric age group(in our study only 2 patients of the 50 patients had ophthalmoplegic migraine).The mean age of presentation was 5 years. The incidence of the disease was less according to our study as well as the literature. The response to steroids proved that ophthalmoplegic migraine is an inflammatory demyelinating neuropathy.

PART 3

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CASE PROFORMA

NAME

AGE:

SEX: MALE/FEMALE

OP/IP NO:

ADDRESS:

PRESENTING COMPLAINTS:

Headache, defective vision, drooping of upper lid (ophthalmoplegic migraine)

HISTORY OF PRESENTING ILLNESS

1.HEADACHE:

Duration, onset, type any radiation, any associated visual phenomena, any nausea or vomiting

2.VISUAL PHENOMENA:

Scintillationscotoma, fortification spectra, micropsia, macropsia, metamorphopsia, pallinopsia, cerebral polyopia, achromatopsia, visual snow, Alice in Wonderland syndrome

3.ASSOCIATED VISUAL FEATURES

Photophobia, negative symptoms(field loss, tunnel vision)

4.DROOPING OF UPPER LID(IN OPHTHALMOPLEGIC MIGRAINE)

Duration, onset, any diurnal variations or fatiguability

5.DEFECTIVE VISION(IN OPHTHALMOPLEGIC MIGRAINE)

PAST HISTORY

Diabetes, hypertension, bronchial asthma, COPD, coronary artery disease, tuberculosis, renal disorder, thyroid disorder, drug abuse

PERSONAL HISTORY

Smoking, alcoholism, diet intake

FAMILY HISTORY

Family history of migraine is asked

GENERAL EXAMINATION

OCULAR EXAMINATION

RE LE

LIDS:

Ptosis (ophthalmoplegic migraine)

Edema (autonomic features of migraine)

CONJUNCTIVA

Congestion (autonomic features of migraine)

CORNEA

ANTERIOR CHAMBER

IRIS

RE LE

PUPIL
Size, shape
Reaction to light
(important in ophthalmoplegic migraine)

LENS

EXTRAOCULAR MOVEMENTS

OCULAR INVESTIGATIONS

VISUAL ACUITY

REFRACTION

FUNDUS(DIRECT,INDIRECT OPHTHALMOSCOPY)

AUTOMATED PERIMETRY

OPTICAL COHERENCE TOMOGRAPHY(RNFL THICKNESS REPORTS)

ADDITIONAL INVESTIGATIONS IN OPHTHALMOPLEGIC MIGRAINE

1.Baseline

2.Thyroid profile

3.CSF analysis

4.Neuroimaging

5.Vasculitis work up

6.Metabolic parameters

7.Others like complement factors,echocardiogram

DIPLOPIA CHARTING(OPHTHALMOPLEGIC MIGRAINE)

ORTHOPTIC EVALUATION(OPHTHALMOPLEGIC MIGRAINE)

MASTER CHART

S.NO	PATIENT NAME	AGE	SEX	DURATION OF MIGRAINE	TREATMENT	VISUAL PHENOMENA	ASSOCIATED VISUAL FEATURES	REFRACTION	TN/RE & LE	COLOUR VISION	FUNDUS	GONIOSCOPY	FIELDS	OCT
1	KALA	40	F	8Y	PAP	S			12 & 14	N	N	O	N	1,N,N
2	POOGAVANAM	34	F	15Y	PAP				2 0 & 20	N	N	O	N	1,N,T
3	PREMAVATHY	30	F	5Y	PAP				12 & 14	N	N	O	N	
4	JOHNSON	40	M	15Y	PAP			NV	20 & 14	N	N	O	N	1,N,N
5	SARASWATHY	36	F	5Y	AP				12 & 20	N	N	O	N	1,N,T
6	SARALA	35	F	7Y	PAP				14 & 20	N	N	O	N	3,S
7	RAJESWARI	49	F	20Y	PAP			NV	10 & 20	N	N	O	N	1,T,N
8	LAKSHMI	38	F	4Y	PA	S		NV	14 & 20	N	N	O	N	1,N,N
9	PARANTHAMAN	46	M	5Y	PA	MM		NV	22 & 14	N	N	O	N	1,T,N
10	NATARAJ	45	M	5Y	AP			NV	12 & 16	N	N	O	N	3,I
11	SHAKELA	55	F	6Y	AP			NV	20 & 16	N	N	O	N	1,N,T
12	FIROZBANU	28	F	2Y	AP		P		14 & 12	N	N	O	N	1,T,I
13	JAYANTHI	26	F	5Y	AP		P		12 & 16	N	N	O	N	1,N,T
14	BALAMANI	49	F	6Y	PAP			NV	20 & 20	N	N	O	N	3,T
15	PARIMALA	37	F	7Y	PAP				20 & 16	N	N	O	N	1,N,T
16	SHANMUGAM	59	M	10Y	PAP			NV	10 & 20	N	N	O	N	1,N,T
17	SIVAGAMI	40	F	5Y	PAP			NV	20 & 14	N	N	O	N	3,N,T
18	JAHEEDA	38	F	5Y	PAP				20 & 14	N	N	O	N	1,T,I
19	GOVINDAMMAL	56	F	10Y	PAP				20 & 20	N	N	O	N	1,T,S
20	LAKSHMI	75	F	9Y	PAP			NV	20 & 12	N	N	O	N	1,T,T
21	THAMAYANDHI	27	F	5Y	PAP	AS			20 & 20	N	N	O	N	2,S
22	SARAWATHY	52	F	5Y	PAP			NV	12 & 18	N	N	O	N	1,N,N
23	MARINA	41	F	10Y	PAP	AS		NV	14 & 18	N	N	O	N	1,N,T
24	PARAMESWARI	43	F	5Y	PAP			NV	14 & 14	N	N	O	N	1,N,N
25	VALLI	40	F	6Y	PAP	Mi		NV	20 & 12	N	N	O	N	2,N
26	CHINTHAMANI	43	F	7Y	PAP		P	NV	16 & 14	N	N	O	N	2,T
27	LAKSHMI	66	F	5Y	PAP		P	NV	16 & 18	N	N	O	N	1,T,T
28	MAHESH	27	M	6Y	PAP	CO			20 & 18	N	N	O	N	4

29	LOGESWARI	32	F	7Y	AP				12 & 18	N	N	O	N	1,T,T
30	RAMAKRISHNAN	37	M	5Y	PAP	Mi	P	NV	14 & 14	N	N	O	N	1,T,T
31	DHARINI	45	F	7Y	PA			NV	16 & 12	N	N	O	N	1,T,T
32	SHREE VIDHYA	33	F	5Y	PAP				16 & 18	N	N	O	N	1,T,N
33	ARJUNAN	48	M	10Y	AP	S	P	NV	20 & 18	N	N	O	N	
34	RAJENDRAN	31	M	10Y	PAP	P			20 & 16	N	N	O	N	1,T,N
35	SUMANGALI	42	F	5Y			P	NV	14 & 18	N	N	O	N	
36	PRABHU	37	M	7Y					14 & 18	N	N	O	N	
37	SAROJINI	31	F	7Y		F			16 & 18	N	N	O	N	
38	ARPUDHA MARY	38	F	8Y			P		20 & 16	N	N	O	N	
39	MAHENDRAN	43	M	10Y		VS		NV	20 & 16	N	N	O	N	
40	RAGUNATHAN	46	M	12Y			P	NV	14 & 18	N	N	O	N	
41	AYESHA BEEVI	48	F	15Y		MM		NV	20 & 18	N	N	O	N	1,T,T
42	SHAHUL AHMED	33	M	10Y		FV			14 & 16	N	N	O	N	2,S
43	BANUMATHI	49	F	15Y				NV	20 & 16	N	N	O	N	
44	PRIYADARSHINI	36	F	6Y				NV	20 & 12	N	N	O	N	
45	VARADARAJAN	41	M	10Y				NV	16 & 12	N	N	O	N	
46	JANAKI	43	F	5Y					14 & 10	N	N	O	N	
47	BHARATHI RAJ	36	M	10Y					14 & 10	N	N	O	N	
48	RANI	38	F	8Y		P				N	N	O	N	

S.NO	PATIENT NAME (OPHTHALMOPLEGIC MIGRAINE)	AGE	SEX	DURATION OF MIGRAINE	TREATMENT	EOM	DIPLOPIA	REFRACTION	TN /RE & LE	COLOUR VISION	FUNDUS	GONIOSCOPY	FIELDS	ORTHOPTICS
1	RISWANA BEGUM	5	F	3Y	SPAP	A,E,D,EX	CD	6/24,6/9	10 & 12	N	N	O	N	IS
2	LAKSHMI	5	F	1Y	SPAP	A,E,D,EX	CD	6/6,6/12	12 & 14	N	N	O	N	IS

KEY TO MASTER CHART

NAME

AGE

SEX

M - Male

F - Female

DURATION OF MIGRAINE

Number

y - years

m - months

d - days

VISUAL PHENOMENA

Fortification spectra - F

Scintillation scotoma - S

Micropsia - Mi

Macropsia - Ma

Metamorphopsia - MM

Pallinopsia - P

“Alice in wonderland syndrome” - AS

Cerebral polyopia - C

Achromatopsia - A

Visual snow - V S

Kaleidoscope vision - KV

Fragmented vision - FV

Mosaic vision - MV

REFRACTION

NV - Near vision add for the patient

ASSOCIATED VISUAL FEATURES

Photophobia - P

Negative symptoms - N

COLOUR VISION

N - Normal

Abn - Abnormal

GONIOSCOPY

O - Open angles

C - Closed angles

IOP

Numbers indicate the measurement in mmHg

FUNDUS

N - normal

Abn - abnormal

OCT

RNFL THINNING

BE - 1

RE ALONE - 2

LE ALONE - 3

BE MULTIPLE QUADRANT THINNING:4

Superior quadrant thinning - S

Inferior quadrant thinning - I

Nasal quadrant thinning - N

Temporal quadrant thinning - T

If BE involved, RE finding first followed by LE

TREATMENT

Steroids - S

Propranalol - P

Amitryptilline - A

Paracetemol - P

DIPLOPIA

UD - Uncrossed diplopia

CD - Crossed diplopia

EOM_Extraocular movements

A - adduction restriction

E - Elevation restriction

D - Depression restriction

EX - Extorsion restriction

ORTHOPTICS

IS - incomitant squint

CS - concomitant squint